THE
BRITISH JOURNAL OF SURGERY

VOL. XXII JULY, 1934 No. 85

# *IPSISSIMA VERBA*

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## II. JOHN HUNTER'S EXPERIMENT

EVERY surgeon knows that John Hunter based his teaching that syphilis and gonorrhœa were manifestations of a single virus and thereby threw back the scientific knowledge of both diseases for nearly a century. Few surgeons have read the account of the disastrous experiment on himself. It is contained in Part vi, Chapter 2, Section ii, p. 324, of the *Treatise on the Venereal Disease* (London, 1786), and is in the following words "*of the Lues Venerea*":—

"To ascertain several facts relative to the venereal disease the following experiments were made. They were begun in May 1767.

"Two punctures were made on the penis with a lancet dipped in venereal matter from a gonorrhœa; one puncture was on the glans, the other on the prepuce.

"This was on a Friday*; on the Sunday following, there was a teasing itching in those parts which lasted till the Tuesday following. In the meantime these parts being often examined, there seemed to be a greater redness and moisture than usual, which was imputed to the parts being rubbed. Upon the Tuesday morning the parts of the prepuce where the puncture had been made were redder, thickened and had formed a speck; by the Tuesday following the speck had increased and discharged some matter, and there seemed to be a little pouting of the lips of the urethra, also a sensation in it in making water, so that a discharge was expected from it; the speck was now touched with lunar caustic and afterwards dressed with calomel ointment. On Saturday morning the slough came off, and it was again touched, and another slough came off on the Monday following. The preceding night the glans had itched a good deal, and on Tuesday a white speck was observed where the puncture had been made; this speck when examined was found to be a pimple full of yellowish matter. This was now touched with the caustic, and dressed as the former. On the Wednesday the sore on the prepuce was yellow, and was therefore again touched with caustic. On the Friday both sloughs came off, and the sore on the prepuce looked red, and it's basis not so hard; but on the Saturday it did not look quite so well, and was touched again; and when that went off it was allowed to heal up, as also the other, which left a dent in the

* The Fridays in May, 1767, were the 1st, 8th, 15th, 22nd, and 29th.

glans. This dent on the glans was filled up in some months, but for a considerable time it had a bluish cast.

"Four months afterwards the chancre on the prepuce broke out again, and very stimulating applications were tried; but these seemed not to agree with it, and by letting it alone it healed up. This it did several times afterwards, but always healed up of itself. That on the glans never did break out, and herein also it differed from the other.

"While the sores remained on the prepuce and glans, a swelling took place in one of the glands of the right groin. I had for some time conceived an idea that the most effectual way to put back a bubo was to rub in mercury on that leg and thigh, which would send a current of mercury through the inflamed gland; this afforded a good opportunity of making the experiment. I had often succeeded in this way, but now wanted to put it more critically to the test.* The sores upon the penis were healed before the reduction of the bubo was attempted. A few days after beginning the mercury in this method the gland subsided considerably. It was then left off for the intention was not to cure it completely at present. The gland some time after began to swell again, and as much mercury was rubbed in as appeared to be sufficient for the entire reduction of the gland; but it was meant to do no more than to cure the gland locally, without giving enough to prevent the constitution from being contaminated.

"About two months after the last attack of the bubo, a little sharp pricking pain was felt in one of the tonsils in swallowing anything; and on inspection a small ulcer was found, which was allowed to go on till the nature of it was ascertained and then recourse was had to mercury. The mercury was thrown in by the same leg and thigh as before, to secure the gland more effectually, although that was not now probably necessary.

"As soon as the ulcer was skinned over the mercury was left off, it not being intended to destroy the poison, but to observe what parts it would next affect. About three months after, copper-coloured blotches broke out on the skin, and the former ulcer returned in the tonsil. Mercury was now applied the second time for those effects of the poison from the constitution, but still only with a view to palliate.

"It was left off a second time, and the attention was given to mark where it would break out next, but it returned again in the same parts. It not appearing that any further knowledge was to be procured by only palliating the disease a fourth time in the tonsil, and a third time in the skin, mercury was now taken in a sufficient quantity, and for a proper time to complete the cure.

"The time the experiments took up, from the first insertion to the complete cure, was about three years.

"The above case is only uncommon in the mode of contracting the disease, . . . It proves that matter from a gonorrhœa will produce chancres.

"It makes it probable that the glans does not admit the venereal irritation so quickly as the prepuce. The chancre on the prepuce inflamed and suppurated in somewhat more then three days, and that on the glans in about ten."

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* The practice in 1767 was to apply a mercurial plaister on the part, or to rub in mercurial ointment on the part, which could hardly act by any other power than sympathy. [Hunter's footnote.]

Although the experiment was begun in 1767 it was not until 1786 that Hunter published this account of it in the *Treatise on the Venereal Disease*. It is remarkable that so acute an observer should have considered himself cured and did not connect his subsequent ill health with a persistence of the disease. The term 'Hunterian chancre' or 'hard sore' soon came into general use in England, though his views were not accepted universally. Richard Carmichael as early as 1814 was teaching that there were different kinds of venereal disease, that they were produced by different poisons, and that they did not all require mercury for their cure. Philippe Ricord repeated Hunter's experiment in 1836 and showed that gonorrhœa was distinct from syphilis. Bassereau in 1852 and Clerc in 1854 taught that soft sores were not due to the same poison as that which produced gonorrhœa or syphilis. It was not until the birth of bacteriology that the age-long controversy was at last ended. In 1879 Albert Neisser, of Breslau, recognized the gonococcus; in 1889 Ducrey announced the discovery of a causative bacillus in pus taken from a bubo associated with soft sores, and on March 3, 1905, Fritz Schaudinn pointed out to Paul Erich Hoffmann the *Spirochæta pallida* as the infecting organism of syphilis.

# THE EXAMINATION OF FRESH TISSUES BY THE WET-FILM METHOD

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THE use of wet films as a method of pathological investigation was introduced by Dudgeon and Patrick[1] in a paper published by them in the BRITISH JOURNAL OF SURGERY in 1927. A method of rapid diagnosis of new growths had previously been perfected by E. H. Shaw,[2] and in his hands the results had been successful. The wet-film method was tried in a series of 200 consecutive cases of new growths and inflammatory tissues, and it was found that not only was it possible to make a complete and accurate diagnosis on the films, but that the specimens obtained were so perfect that the details of cell structure could be appreciated in a way not possible in the ordinary section. Encouraged by these results the work has been continued since that time, and this paper represents an analysis of over 1000 cases.

Dudgeon and Patrick emphasized the value of wet films as follows: "we consider the advantages are the beautiful preparation of individual cells and fragments of tissue which are seen in the films, the simplicity of the technique, and the small amount of material required for the preparation of the films." The additional experience gained since that time has confirmed these views, and it is suggested that the practical value of the method is much greater than is generally realized. It may be argued that the existing methods are sufficient for the purpose, but one cannot fail to be impressed by the possibilities which present themselves and which are not open, for one reason or another, to these methods.

The fixing of the wet cells with Schaudinn's fluid is very satisfactory and they are so little damaged by the process that they are presented as nearly as possible in their true form.

A very wide area of the original tissue can be covered. This point is of importance, for not only does it enable the pathologist to study normal and abnormal cells in the same preparation, but in those conditions where differences of histological structure occur in various parts of a specimen, they are more likely to be represented in a wet film than in a section taken from a small area.

It must be emphasized that the method has been found to be applicable to all types of lesion, and although it was originally devised as a way of obtaining a rapid diagnosis in the case of tumours it has since been used for the more detailed study of every tissue which could be obtained in a fresh state.

The study of tissues as a whole has been shown to be of great interest, and in the later part of this investigation the routine of making wet films from tissues far removed from the main lesion was adopted. In the breast, for instance, where radical amputation had been done for carcinoma, wet films were made from the

muscle, the skin, and outlying parts of the gland, and it was found that in a large percentage of cases malignant cells were discovered lying amongst the normal tissues. *Table I* gives the actual results of this investigation.

*Table I*

| Origin of Tissue | Cases Examined | Carcinoma Cells found in Apparently Normal Tissues |
|---|---|---|
| Breast .. .. | 37 | 26 |
| Stomach .. | 8 | 3 |
| Rectum and colon | 9 | 6 |
| Uterus .. .. | 3 | 3 |
| Tongue .. .. | 1 | 1 |
| Total .. | 58 | 39 |

It will be seen that the total number of cases investigated was 58, and in 39 (67·2 per cent) carcinoma was found in apparently normal tissues which had not reacted to its presence. The malignant cells were often isolated from each other or in small clusters, but they differed so markedly in their appearance and behaviour from normal cells that the diagnosis could be made with certainty. By this method, therefore, not only can the spread of disease be investigated, but a more accurate judgement as to the extent of a lesion can be formed.

In the operating-theatre most surgeons form their opinions on the macroscopic appearances of the conditions they meet; but in any case of doubt, where the immediate treatment depends upon an exact diagnosis, it is well to remember that this can be given in ten minutes, and it is as exact in its rendering as the more prolonged investigation by means of sections. As an example of the value of a wet film to the surgeon the case quoted by Wrigley[7] is interesting. The conclusion had been reached that a certain abdominal tumour was malignant, but a wet film was taken and the diagnosis of cystadenoma of the ovary given immediately. The operation was therefore continued and the tumour was successfully removed.

The materials necessary for making wet films can be taken from place to place in a very small box, and the method has been used successfully on many occasions away from the laboratory. In the out-patient department and wards of a hospital or a consulting-room it can be applied particularly to the diagnosis of doubtful lesions of the skin and exposed parts of the body, and consequently loss of time may be avoided, a diagnosis can be given immediately, and appropriate treatment can be instituted.

It remains to be said that with these advantages there are certain limitations. There is no doubt that a sound knowledge of pathological processes, as they are seen in ordinary sections, is required, and to this must be added a great deal of practice in studying wet films made not only from diseased areas, but from the normal tissues in the vicinity. Again, the practical difficulty of obtaining the

material in a completely fresh condition is very real, and the specimens must not be put into water, saline, or preservatives.

## TECHNIQUE

The method which has been used is practically the same as that described in the original paper.[1] It has been emphasized that the correct interpretation of a wet film depends on the perfect presentation of the individual cells, and this can best be obtained by observing the following technique, the minor points of which are discussed in detail. The actual scraping of the tissue needs careful handling. Absolutely fresh material is essential. Water, saline, preservative, or fixing solution makes the tissue useless for wet films. The same is true of post-mortem material, for in this the earliest changes of autolysis are sufficient to distort the normal appearances and make diagnosis difficult or impossible.

Take the specimen, therefore, as soon as it has been removed by the surgeon and examine it carefully with the naked eye to determine the part most affected by the disease. This should present but little difficulty, and yet it is surprising how often a film can be made from a normal area in the vicinity of the lesion or from one in which autolysis or degeneration is so far advanced that the cells cannot be recognized. As an illustration of this practical point the following case is instructive. A patient attended the Out-patient Department complaining of a chronic ulcer on the dorsum of the hand; as the diagnosis was in doubt a wet film was prepared from the surface of the lesion, and on examination this showed acute inflammation and masses of degenerating surface epithelium. A second film made from a small piece of tissue removed under local anæsthesia from the edge of the ulcer revealed a squamous-cell carcinoma. Particular care is necessary in the case of films made from lymphatic glands.

It is well to dry the tissue with a piece of gauze, for the red blood-cells which are certain to be present in large numbers are not needed in the preparation, and the tissue fluids which exude from the cut surface upset the evenness of the film by lying over the individual cells and producing unequal staining.

The film is made by scraping the tissue with a sharp knife so that the cells are collected upon the blade and can be transferred immediately to a glass slide. A smear is then made in the same way as a blood-film.

The optimum thickness is a matter of experience, but on the whole films of very cellular structures, such as lymphatic glands or malignant growths, should be thin, whereas in the case of chronic mastitis, for instance, where the acini are few and far between, a wide area must be covered and a fairly thick film prepared. Once the film has been made, no time must be wasted in removing from the slide those parts which are too thick or lumpy, for it is essential that it be placed in Schaudinn's fluid immediately, before drying occurs.

This fluid is prepared by adding one volume of absolute alcohol to two volumes of a saturated aqueous solution of mercuric chloride. The mixture can be kept indefinitely as a stock solution, but immediately before use glacial acetic acid to the strength of 3 per cent must be added.[4] If the acid is added to the stock solution a long time before it is used for fixing the wet films, it will be found that there is a tendency for crystallization to occur. The best fixation is achieved by leaving the slide immersed in this fluid for as long as twenty minutes;

when, however, a rapid diagnosis is needed a minimum of two minutes is all that is necessary. The slide is then transferred to methylated spirit to which has been added a few drops of tincture of iodine. The iodine has the property of removing the excess of mercuric chloride by converting it into the more soluble mercuric iodide. A washing in distilled water prepares the slide for staining.

It has been found that Mayer's hæmalum gives the most constant results as a nuclear stain. It is made by dissolving 1 grm. of hæmatoxylin in 1000 c.c. of distilled water. The solution must be heated to get the hæmatoxylin to dissolve properly. To this are added 0·2 grm. of sodium iodate, 50 grm. of ammonia alum, and finally 20 c.c. of glacial acetic acid. The advantages of this particular stain are that it is much cheaper than the hæmalum which is made from hæmatein, and the sodium iodate enables it to be used immediately without having to wait for 'ripening' to take place. The slide is left in the stain for about two minutes or less, depending upon the strength of the solution. It is then transferred to tap water. In the water the colour changes from a reddish purple to blue, and the slide must be left until 'blueing' has taken place. During this time the thick or uneven parts of the film can be removed from the slide by touching them with a needle. As a counterstain a weak solution of eosin is used.

The slide is then taken through the alcohols to xylol in the usual way and mounted with Canada balsam and a cover slip.

Several stains besides hæmalum and eosin have been tried, but we have not found any which bring out the important features in a way superior to these, and only in special cases has it been necessary to use other methods.

## POINTS IN THE INTERPRETATION OF WET FILMS

Some account of the various pictures presented by the more common pathological conditions will now be given.

Normal epithelium presents a most perfect picture. The cells are seen either in flat plaques like the stones of a finely tessellated floor, as individual cells, or as groups lying side to side. They are easily recognized since the staining reaction and general outlay are remarkably constant. The only exception to this are the cells found in the superficial strata of desquamating squamous epithelium, and these are often irregular in shape and separate from each other. On close examination of normal epithelium the following points can be observed: the similarity in size and shape of the cells; the fact that the nuclei are placed in like positions with regard to the remainder of the structure; the very fine reticular arrangement of the nuclear chromatin, giving the appearance of even consistency to each nucleus; and finally the tiny pink nucleoli. Further, it will be seen that where several cells are joined together in a plaque the cytoplasm stains differently towards the margin of each cell, for whereas in the main body of the cell it is blue or purple in colour, towards the edge there is a lighter area, which is itself bounded by a fine dark line. It is this dark line, lying as a sort of fence between adjacent cells, which is responsible for the tessellated appearance described above. Isolated cells appear to be limited by a definite cell membrane.

In the plaques the outline of the cells is angular and they fit against one another like the chambers of a honeycomb, and the edges may show nuclei and protoplasm extruded leaving delicate fringes of the ruptured cell membranes.

It is interesting to note that Shattock and Dudgeon,[3] in a paper read before the Pathological Section of the Royal Society of Medicine in 1914, included a diagram which was described as follows: "A plaque of carcinomatous cells detached from one of the intact eminences of a tumour of the colon by lightly rubbing a cover-glass over it, examined in glycerine . . ." The method, it will be noted, is not that which is described in this paper. The diagram actually shows a plaque of perfectly normal epithelium and demonstrates the characteristic features mentioned above. It shows incidentally how easily a piece of tissue can be taken from the wrong place. In this case it was assumed that the cells must be malignant in view of the site from which they were thought to have come.

The stroma of a tissue is represented, but the cells are not easy to differentiate, and except in the case of some malignant growths they are of limited interest. Arteries and veins cannot be distinguished as such, but capillaries are sometimes beautifully shown, and these have been observed to perfection in many tissues, and were specially noticed in gliomata and lesions of the central nervous system.

To this picture of normal cells the features of inflammation may be added. The polymorphs of acute inflammation are particularly easy to identify, for they are large and natural as in a blood-film. The same applies to the large mononuclear cells, plasma cells, and lymphocytes of chronic inflammation.

The phagocytic properties of polymorphs, large mononuclear cells, and malignant cells of all types have been repeatedly observed — special reference to the malignant cells will be made in another section of this paper—and the various types of giant cells which are typical of special conditions can be quickly recognized.

As an example of the application of these points a wet film made from a small tumour in the subcutaneous tissues of a patient's leg may be quoted. It showed a large number of foam cells, many of which were packed with pigment, masses of small round cells, and giant cells of the foreign-body type. A second film was made from the tumour and stained to show free iron, and this being positive a diagnosis of organizing hæmatoma was correctly given. It may be remarked that in cases of doubt as to the nature of a pigment, melanin can be quickly distinguished from blood pigment, since it does not give the free iron reaction.

Normal epithelium can be modified considerably by the presence of acute inflammation. In this condition the cells may be large and more swollen than normal, the boundaries less distinct, and the cells themselves separate from one another. In addition there may be variations in the size and shape of the nuclei. Some of these features are due to degeneration, and this is shown by pink staining of the cytoplasm. Close inspection, however, reveals the fine reticular arrangement of the nuclear chromatin, and remnants of the tessellated plaques will nearly always be found. In these cases it is particularly the degenerating individual cells which are apt to be misleading.

The features of tuberculosis are readily defined. In support of this it can be said that the diagnosis has been made from the wet film when this was not possible from the corresponding section, and the accuracy of the verdict has been upheld by the subsequent clinical progress of the patient.

Lymphadenoma is easy to diagnose in wet films, and the 'rapid technique' is particularly useful in cases where the diagnosis has to be made from tuberculous adenitis, for the surgical treatment of the glands is different in the two conditions.

There is little to say as regards the distinctive features of benign neoplasms except that in these tumours all the characters of normal cells of the same tissue are shown and that there are usually more cells in the field. A polyp of the rectum, for instance, shows more epithelial cells and less stroma than a piece of rectal wall. Since, however, this distinction is only relative, it cannot be held to be characteristic of benign tumours.

In the diagnosis of wet films made from tumours the main point at issue is the simple or malignant nature of the cells. Now it has always been strongly held that "in the appearance of a cell from a cancer there is nothing characteristic of the disease, nothing that would lead a pathologist to identify it as a malignant cell. Cancer can only be identified in sections showing the relation of cells to each other in a group." This is the view expressed by Bland-Sutton[5] in 1922, and it is evident that it still holds the field, for in 1932 Ludford[6] writes: "an extensive search has been made for any difference between normal and malignant cells which might help to explain the cause of malignant growth, or reveal some weak spot in the armour of the cancer cell towards which therapeutic attack might be directed. It has been pointed out that we have no definite morphological criterion of malignancy, but certain differences between normal and malignant cells have been established." He goes on to say that these differences are concerned with the reaction of malignant cells towards acid vital dyes.

From the study of wet films, it seems certain that malignant cells are different in appearance from normal cells and that their special histological features are so striking that they can be picked out as isolated units in a large field. The truth of this statement is borne out by the following figures from the series under consideration.

| | |
|---|---|
| Total number of wet films of malignant disease examined .. | 469 |
| Total number diagnosed as malignant .. .. .. | 462 |

It is not suggested that a solitary cell could be pronounced malignant on its own merits, but in a field where benign cells are present for comparison the diagnosis is not difficult. It depends upon the special characters of the individual cells and not upon the presence or absence of invasion of normal tissue, although if this point is shown in the wet film it is taken as corroborative evidence. The features which in a wet film suggest that a cell or group of cells is malignant can be defined as follows:—

Malignant cells stain more deeply than their benign prototype. This is due to the fact that the nuclei have a greater affinity for hæmalum. They vary in size and shape, they are larger than normal, and their position relative to the surrounding cytoplasm is in no way constant. The nuclear chromatin is arranged in thick, irregular, and deeply staining bundles, and the fine reticular arrangement of normal cells is rarely seen. The nucleoli are much larger than normal, they are sometimes multiple, and stand out as purple or pink dots in the sphere of the nucleus.

Mitosis is beautifully demonstrated, but it cannot be regarded as characteristic of malignant cells alone. It has been observed in many benign conditions, and all that can be said is that it is most frequently found in malignant cells. It is worth noting, however, that in tumours such as papillomata of the urinary system, which frequently prove to be malignant, mitosis is very much in evidence. Large

numbers of mitotic figures are presumably an indication of the rapid proliferation of the cells concerned, but the cases of carcinoma of the breast which were followed up in detail and which are reported in this paper showed quite definitely that the picture was not necessarily of bad prognostic significance.

The actual size of the cells varies enormously, so that extremely large and very small ones can be found lying side by side in the same preparation. The larger cells are often multinuclear.

Malignant epithelial cells do not stick together in regular formation, and placards are the exception rather than the rule. In those cases where placards are found the cell boundaries are usually not apparent and the appearance is that of a syncytium from the edges of which nuclei, devoid of cytoplasm, can be seen breaking away. In some films, however, the cell boundaries are prominent, and in spite of this the cells separate from each other. This was particularly noticed in a film made from a carcinoma of the stomach, in which the boundaries were very obvious and yet the majority of the cells were isolated from each other; several placards were present, but these were different from those of benign epithelium, for the regular geometrical arrangement was lacking.

It is a feature of benign epithelium that the sheets of cells are not usually more than one layer in thickness, and in films of malignant tissue the appearance of placards is often due to the cells lying on top of each other in heaps. In normal epithelium the cells stick together because there is some cement substance between the individual units. This substance is capable of holding them together by itself, for in wet films made from normal epithelium many cells are presented in rows and it is easy to see that there is no basement membrane supporting them. Now there is evidence that the cement substance is either not represented in malignant tissue or differs in nature from that of benign epithelium, for malignant cells separate very completely from each other and many are disrupted so that the nuclei are entirely devoid of surrounding cytoplasm.

In malignant growths there are numbers of degenerating cells. In the early stages they are swollen and the outlines are distorted by alterations in the structure of the cytoplasm, which stains more pink as the condition advances. The nuclei, which at first are very large, gradually shrink and the chromatin condenses into a solid mass which ultimately breaks up and disperses in many basophil granules. In some cases the nuclear membrane is destroyed before the cell disintegrates and small discrete blocks of chromatin can be seen lying at random in the cytoplasm. Inclusion bodies are found in all types of malignant cell, but are most prominent in epithelial tumours.

It has been pointed out by Carleton[8] that "it is usual to regard phagocytosis as the 'perquisite' of various motile cells which are generally regarded as mesodermal in origin", but that his conclusions lead him to suppose that "certain mammalian epithelia are capable on appropriate stimulation of responding by phagocytosis to the call made upon them." Phagocytosis is therefore not commonly seen in benign epithelium except under special conditions, and never in the same rampant form as in malignant epithelial cells. The special powers of phagocytosis possessed by malignant cells have been mentioned already. It is often surprising how much material can be taken up by a single cell, for it is not unusual to find many polymorphs in the cytoplasm. Phagocytosis appears to be most in evidence in those tumours which have been submitted to a course of radium or X-ray

treatment, and in the tumour cells polymorphs, red blood-corpuscles undergoing hæmolysis, tissue cells, pigment, and débris can all be seen.

With these points in view it is often possible to give an opinion not only as to the malignant nature of a group of epithelial cells but also on the type of epithelium from which they originated. Great variation exists, however, and sometimes the finer points such as the difference between a columnar- and a spheroidal-cell carcinoma cannot be determined. The absence of stroma in wet films of carcinoma may lead to difficulty in distinguishing the condition from sarcoma, especially as the amount of cytoplasm in the cells cannot be taken as a guide one way or the other. Sarcoma cells show all the abnormalities of nuclear structure which are found in carcinoma but often to an exaggerated degree. The separation of the cells from each other is more marked, and there is seldom any clue in the wet film as to the origin of the tissue. In pigmented tumours the blood pigment must be distinguished from melanin.

There remain for consideration the rodent ulcers which are locally malignant, and here the characteristics of the film histology are intermediate between those of a carcinoma and a simple tumour.

## SPECIAL SYSTEMS

In the following account the special points which have arisen during the course of this investigation are set out and discussed in detail under the appropriate organs or systems whence the specimens were obtained.

**Alimentary System.**—The value of wet films has been particularly evident in the diagnosis of the various pathological conditions of the lips, mouth, and tonsils. A small piece of tissue can easily be removed under a local anæsthetic, and the diagnosis can often be determined at once. This has been done with success in many cases, and of these the following are quoted as examples. A patient came to the Hospital complaining of a large, painless, and punched-out ulcer on the dorsum of the tongue. The ulcer was judged to be a gumma which had broken down, or a carcinoma. The Wassermann reaction was found to be negative. A wet film had been made, however, and this showed the most perfect endothelial-cell proliferation, caseation, and giant-cell formation, so that a diagnosis of tuberculosis was given. Subsequently the patient was found to have pulmonary tuberculosis, and under appropriate treatment the ulcer healed. Again, a man came to the Out-patient Department complaining of a swelling of the right tonsil. The tonsil, on examination, was slightly bigger than normal, but it was quite soft on palpation and the diagnosis of malignancy was not considered at the time. A wet film was taken. The tumour proved to be an epithelioma and the patient was admitted shortly afterwards. The importance of this case is that in the ordinary course of events the diagnosis would have been in doubt until after admission, probably a month later, and this valuable time would have been wasted.

In wet films from the stomach some of the most interesting features have been found (*Figs.* 1, 2A). There is probably no site in the body, except perhaps the endometrium, the gall-bladder, and the prostate, where the tessellated appearance of benign epithelium is presented so beautifully.

The cases of ulcer and carcinoma of the stomach which came for diagnosis were correctly reported upon in every instance. An interesting observation has

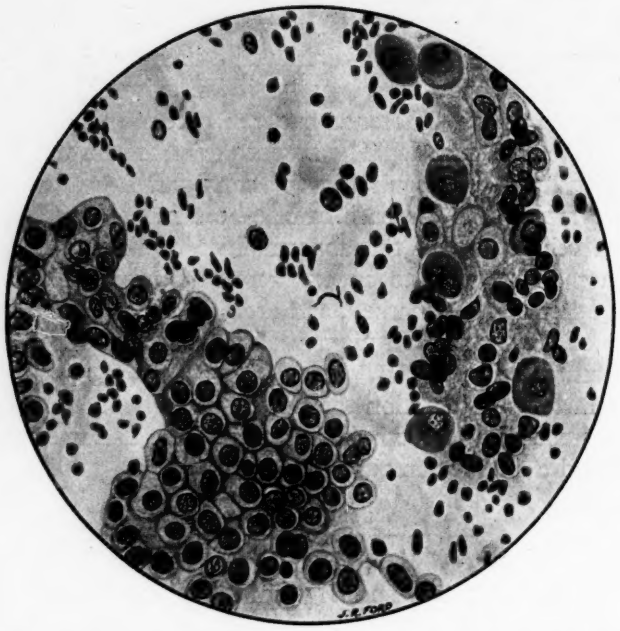

FIG. 1.—Gastric mucosa. Showing placards of normal epithelium with regular cell boundaries on the left of the picture: on the right are the large oxyntic cells. Red cells are scattered throughout the field. (× 300.)

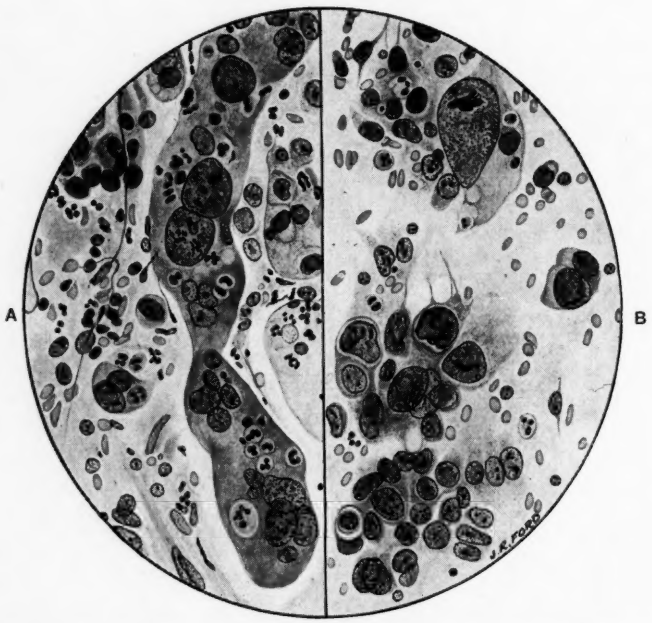


FIG. 2.—A, Stomach. Carcinoma. Note the large syncytial masses of malignant cells. Some of the nuclei are very large and a considerable degree of phagocytosis of polymorphs is shown. B, Bladder. Carcinoma. This diagram shows the variation in the shape and size of the cells, many of which have a large nucleus and very little cytoplasm. (× 300.) (These two figures should be compared with *Figs.* 1 and 3.)

been made with regard to the oxyntic cells which have been supposed to secrete the hydrochloric acid of the gastric juice. These cells are often well seen in a wet film; they are usually arranged in small groups of five or six, and since they are larger than columnar epithelial cells it is easy to pick them out (*Fig.* 1). In addition the nuclei may stain more pink than those of epithelial cells and the cytoplasm contains very fine eosin granules.* In this series there were only 9 cases in which a test-meal had been done before operation, and in these particular cases the presence or absence of oxyntic cells was recorded as shown in *Table II.*

*Table II*

| Diagnosis | Test-meal | Oxyntic Cells |
|---|---|---|
| Sarcoma .. | Total HCl 0·190 | None |
| Carcinoma .. | No HCl<br>Lactic acid + | None |
| Carcinoma .. | No HCl<br>Lactic acid + | Oxyntics + |
| Carcinoma .. | No HCl<br>Lactic acid + | Oxyntics + + |
| Gastric ulcer .. | Total HCl 0·113 | Oxyntics + + |
| Carcinoma .. | No HCl<br>Lactic acid + | None |
| Gastric ulcer .. | Total HCl 0·059 | None |
| Gastric ulcer .. | Total HCl 0·33 | Oxyntics + + |
| Carcinoma .. | No HCl<br>Lactic acid + | Oxyntics + + |

In addition to these there were 11 cases of gastric ulcer, in 10 of which oxyntics were found, and 2 of carcinoma, both showing the same thing. Oxyntic cells were present in films made from the stomach in cases of gastric carcinoma which have no free HCl in the gastric juice.

**Urinary System** (*Figs.* 2B, 3).—Papillomata of the bladder and renal pelvis, which often prove to be malignant, may be diagnosed as carcinoma earlier in a wet film than in a section, and there were two occasions in the series on which this was possible. The diagnosis was subsequently confirmed by the clinical progress of the patients.

The report on a case of hypernephroma gives a good idea of the characteristic appearances of these tumours; wet films were made from two different areas. The ' red ' area showed placards of large cells with small nuclei and clear cytoplasm together with numerous isolated cells of the same type; in addition there were large numbers of hæmolysing red cells. The ' yellow ' area was quite different and

* With higher magnification than has been used for *Fig.* 1 the eosinophil granules of the oxyntic cells can be clearly seen.

presented a solid mass of finely granular foam tissue, throughout which were numbers of solitary nuclei. The outline of the individual cells was indefinite, and the nuclei varied in size and shape.

The secondary deposits from malignant growths of the kidney are often very true to type, so that it is frequently possible to state from a wet film that the malignant epithelium is of the transitional-cell variety. In the following case the surgeon had operated upon a female with a large tender tumour in the upper part of the abdomen on the left side. On inspection of the abdominal viscera a malignant mass was found lying between the stomach, spleen, and kidney; in addition there were two collections of hæmorrhagic exudate, the one in the lesser sac and the other in the transverse mesocolon. It was impossible to state the exact site of the primary tumour, but the posterior wall of the stomach seemed to be the most

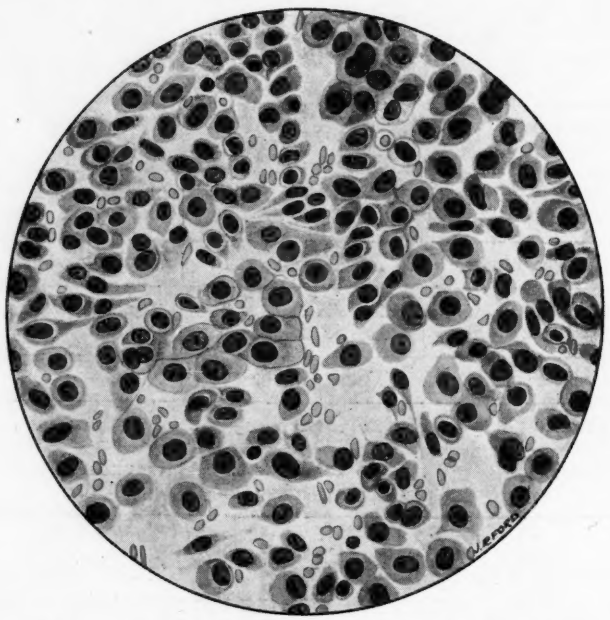

FIG. 3.—Bladder. Papilloma. This diagram shows the regular size of the individual cells, placards of cells, and the abundance of isolated cells, all with a large amount of cytoplasm. (× 300.)

likely place. A small secondary lying in the great omentum was removed for microscopy. Wet films made from this secondary deposit showed a transitional-cell carcinoma, and at the autopsy on the following day the kidney was found to be replaced by a mass of new growth which had started in the pelvis of the kidney and spread into the stomach wall and the spleen.

Benign enlargements of the prostate are very easy to diagnose, for the arrangement of the epithelial cells into placards is clearly shown, the tessellation is obvious even with a low power of the microscope, and the corpora amylacæa are usually plentiful. In the original paper a drawing made from a wet film of an adenoma of the prostate was included. The features are so constant that malignant conditions can be quickly identified from benign. The similarity between sections made from adenoma of the prostate and certain conditions of the breast is well known;

in wet films the two varieties of epithelium are seen to be quite different in appearance, for that of the breast never demonstrates the tessellation to the same degree of perfection as the placards from the prostate.

From the surgical point of view the danger of removing a prostate in the presence of cystitis is recognized, but it has been emphasized in an investigation of the 23 cases in this series in which the history of the progress after operation was available. There were 12 who did not show any evidence of inflammation in the wet film, and 10 of these patients had a normal convalescence; the other 2 died, the one of uræmia and the other of cardiac failure; but in neither was there gross infection in the bladder or kidneys. In the other 11 cases varying degrees of acute inflammation were recorded from the wet film. Two of the patients died from suppuration in the abdominal wall, and the remainder all developed septic complications of varying kinds.

The presence of acute infection in these cases was not sufficiently evident in the corresponding sections for comment to be made upon it.

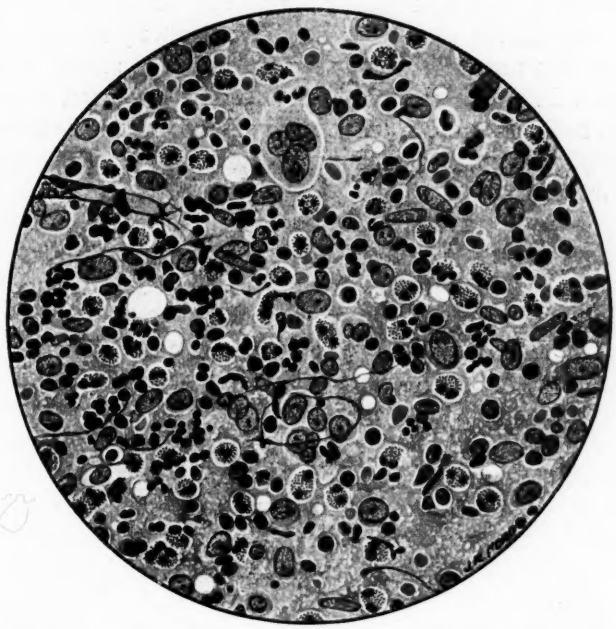

FIG. 4.—Lymph-gland. Lymphadenoma. Showing a considerable eosinophilia. (× 300.)

**Lymphatic System.**—A normal lymph-gland shows a highly cellular field of separate lymphocytes and a smaller number of large mononuclear cells. The nuclei of the lymphocytes, which are remarkably constant in size, shape, and staining reaction, take up the hæmalum well, and the cytoplasm is not shown. The stroma is not represented. It is sometimes difficult in a case of tuberculous lymphadenitis to pick out the best area of gland from which to make a wet film, for one is apt to get too much caseous material and débris by scraping the wall of an abscess cavity, and a film made from a neighbouring gland may show nothing more than chronic lymphadenitis. The best practice, therefore, is to scrape

suspicious patches of the gland and make a film which includes cells from a number of different places.

The picture of lymphadenoma is one of the most satisfactory shown by this method, for in almost every instance the wet film is very much better than the corresponding section. The picture is one of a lymph-gland in which there is evidence of endothelial-cell proliferation; to this are added the two varieties of lymphadenoid giant cell and a number of eosinophils (*Fig.* 4). The cells are all separate and consequently they can be studied individually. In every case of lymphadenoma there have been some eosinophils to be found, and it is worth noting that on the two occasions when a faulty diagnosis was made the wet film presented some of the characters of lymphadenoma but there were no eosinophils. The differential diagnosis rests between chronic lymphadenitis, tuberculosis, and endothelioma of the lymph-gland.

The diagnosis of chronic lymphadenitis is often one of exclusion. The presence of many cells in addition to the lymphocytes suggests some pathological process, but the absence of sufficient evidence pointing towards tuberculosis or other specific condition often necessitates a verdict of chronic lymphadenitis, and it can safely be said that if the smear has been properly made, this diagnosis will be as complete as the diagnosis from the section. The subsequent history of the cases in this series in which chronic lymphadenitis has been diagnosed has been followed up, and, as far as one can tell, these patients have not ultimately developed tuberculosis, lymphadenoma, or malignant disease, so that the true cause of the lesion remains obscure.

**Muscle** (*Fig.* 5), **Connective Tissues, and Bone.**—The majority of lesions in this section have been of a neoplastic nature, but several organizing hæmatomata of muscle and fat have been correctly diagnosed. The only points which arise in connection with sarcomata are that the actual type of cell is often reported upon differently in the wet film and in the section—that is, a tumour in the film may show the features of a round-cell sarcoma, whereas the section may be returned as a fibrosarcoma.

**Central Nervous System.**—Specimens from the central nervous system have not been sufficiently numerous for definite conclusions to be formed, but the fixation of the films in Schaudinn's fluid appears to give valuable information in differentiating the 'gliomata'. An investigation of these tumours was started in conjunction with the late Sir Percy Sargent, but the work has had to be abandoned. Hæmalum and eosin do not bring out the finer points which are claimed for the more complicated methods, though the detail in a wet film is very much greater than that of an ordinary section. A spongioblastoma multiforme was reported upon thus: "A malignant growth with cells of the sarcoma type. Very little cytoplasm around the nuclei. Capillaries beautifully shown. Giant cells and foam cells present. Delicate fibrils can be seen running in all directions from the malignant cells."

**Breast** (*Fig.* 6).—The most important group of wet films which have been made are from the breast. The total number of cases examined was 212, and *Table III* (p. 18) gives the figures in detail.

Normal breast tissue shows a great deal of fat and very little epithelium; in fact, except in the vicinity of the nipple it is quite possible to miss the epithelial elements altogether in a scraping. In lactation, however, large epithelial cells are abundant and these contain the characteristic globules.

FIG. 5.—Muscle. Spindle-cell sarcoma. Note the variation in the size of the cells and the large amount of cytoplasm. (× 300.)

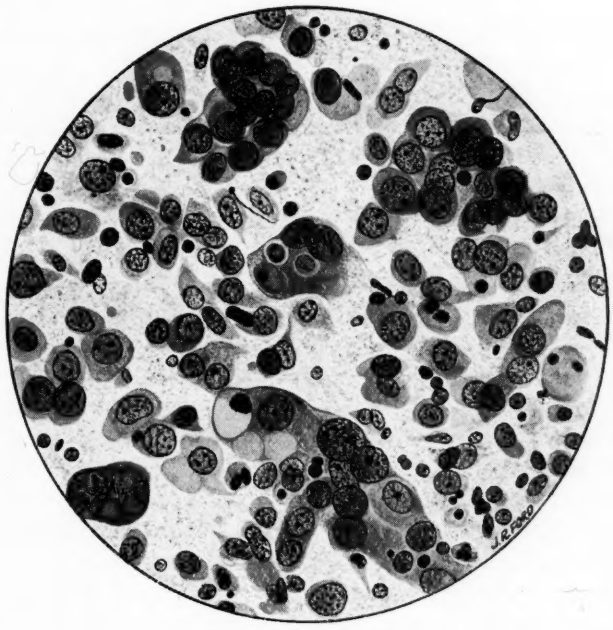

FIG. 6.—Breast. Spheroidal-cell carcinoma showing syncytial masses and large amount of cytoplasm in the cells. (× 300.)

It is sometimes difficult to differentiate a case of chronic mastitis from a normal breast in a wet film; but in the former, although the epithelium is scarce, there is a fibrous-tissue stroma, there are groups of partly degenerate cells from the cyst walls or dilated ducts which stain pink with the eosin and are much larger than the normal epithelium, and a number of large foam cells. A diagnosis of 'simple' as opposed to 'malignant' is the important point in most instances, and this should not be difficult except possibly in the case of proliferative mastitis. Here the epithelial cells are very numerous indeed. They are small, deeply stained, and arranged in placards; but it may not be easy to see the cell boundaries.

*Table III*

| Disease | No. of Cases | Wet-film Diagnosis Inaccurate |
|---|---|---|
| Chronic mastitis<br>Atrophy of breast<br>Galactocele<br>Hæmatoma .. | 41 | 1 diagnosed as carcinoma. The case was actually one of proliferative mastitis |
| Tuberculosis .. | 3 | None |
| Fibro-adenoma .. | 17 | 7—4 cases were diagnosed as carcinoma<br>3 cases were diagnosed as duct papilloma |
| Fibroma .. .. | 3 | 1 diagnosed as a giant-cell sarcoma |
| Duct papilloma .. | 9 | None |
| Sarcoma .. .. | 5 | 1 diagnosed as an inflamed fibroma |
| Carcinoma .. .. | 134 | 2—*a*. "No evidence of malignancy." When re-examined this was obviously wrong and many carcinoma cells were found in the wet film<br>*b*. "No evidence of malignancy." The films were made from the wrong place |

The fact that a galactocœle may simulate tuberculosis both clinically and microscopically was emphasized by the Royal Commission on Tuberculosis. In wet films the special features of the two conditions are clearly demonstrated, and in this series a correct diagnosis has been possible in every case. It will be remembered that chronic inflammation is in evidence in both conditions, but in a galactocœle there is usually some remnant of acute inflammation, and to this are added the typical foreign-body giant cells which are often filled with lipoids. In tuberculosis endothelial cells may be present in large numbers. Foam cells are found in lesions of tlhe breast such as chronic inflammation or mastitis, and it is unusual to discover them in wet films made from malignant growths unless the scraping has included mastitis around the growth, or an area of lactating tissue.

The main point, however, is the nature of the carcinoma cells and their relation to the type of cell found in fibro-adenomata.

It has been noticed that there are three different and distinct types of carcinoma of the breast as presented in wet films. These types depend upon the actual appearance of the malignant cells and are not concerned with the clinical aspect of the tumour.

*Type I.*—In this type the carcinoma cells are very large indeed. Their nuclei are often enormous and the cytoplasm of each individual cell is plentiful. This cytoplasm has a tendency to stain somewhat more pink with eosin than is usual. The cells are arranged in small groups and as isolated units, but there is never any indication of the tessellated appearance of benign placards. In addition multinuclear malignant cells are conspicuous, and these often show phagocytosis of red blood-corpuscles. A diagram of this type of carcinoma was given in the original paper.[1]

*Type II.*—The cells are smaller than those of *Type I*, but are still considerably larger than normal. They have very little cytoplasm and are almost entirely separate from each other; this lack of epithelial formation is suggestive in some cases of a sarcoma. It is in this type that mitosis is especially obvious and the capillaries are sometimes very prominent.

*Type III.*—The cells are small and of about normal size. They are very numerous and arranged in placards; the cell boundaries, however, cannot be seen. There is often evidence of invasion of fat and isolated cells are present.

It is the third type of carcinoma which has led to difficulty in distinguishing between carcinoma and duct papilloma or fibro-adenoma, for in all three the size of the cells is somewhat similar. It will be seen that in a total of 17 fibro-adenomata 4 were judged to be carcinoma of the small-cell type. Having realized that this type of carcinoma is found in wet films one is very much less likely to make the mistake, for the arrangement of the cells in the benign conditions is regular and even, whereas in the carcinoma the nuclei are unevenly spaced and lie upon each other in a characteristic fashion. Corroborative evidence may be found in the presence of isolated cells invading the fat. In a wet film the fat cells tend to lie in plaques like other benign cells and hence the presence of any malignant infiltration is quickly seen.

The significance of the three types of carcinoma cell is difficult to determine, and it was hoped that some light might be thrown upon the problem by studying the after-history of the patients. It has been possible to investigate 129 out of 134 patients, and of these 44 are dead already.

Although the time since operation is small in many of the cases, there is no evidence that any one of the above types is more lethal than another. The patients with the small-cell tumour die or recover in about the same proportion as the others.

With this follow-up available the question of the prognostic significance of mitosis was also investigated, with the same negative result.

**Female Genital System.**—The results of investigations by wet films of lesions of the female genitalia have been studied and reported upon in detail by Wrigley.[7]

A point which might be of use in the early detection of pathological processes in the mucosa of the cervix uteri has been described in a paper by Schiller.[9] It is important to be able to differentiate easily between normal and pathological conditions of the cervix, and it was found that the normal epithelium in this region contains glycogen in its superficial layers, and this glycogen can be demonstrated in the living patient by painting the area with Lugol's iodine solution (iodine, 1; potassium iodide, 2; water, 300). In pathological conditions of the mucosa, the absence of glycogen leaves the cells unstained by the iodine. The diagnosis between leucoplakia and carcinoma in the unstained areas could probably be settled by taking a scraping and making wet films.

## MISTAKES AND DIFFERENCES IN DIAGNOSIS

A critical investigation of the mistakes in diagnosis which have been made in this series confirms the view that with increasing experience these should be reduced to a negligible figure. In the first 200 cases reported by Dudgeon and Patrick there were 9 instances in which the interpretation of the wet films was wrong, and these mistakes have been fully explained in their paper. In the remainder of the series there were only 20 mistakes. The wet films were made from the wrong part of the specimen in 5 of these cases, since the interpretation of the films as they were represented was undoubtedly correct and yet it differed from the section diagnosis. A typical example of this group was a film made from a tumour of the breast in which there was no evidence of neoplasm, but the section showed a cellular carcinoma.

In two films, the one made from a carcinoma of the prostate and the other from a lymphosarcoma of the subcutaneous tissues, a diagnosis should not have been attempted, since the preparations were so bad that the cells could not be recognized distinctly.

Three cases were correctly diagnosed at a second examination of the films, and it is admitted that these mistakes should not have occurred.

The difficulty of differentiating between fibro-adenoma of the breast and the small-cell type of carcinoma has been explained elsewhere.

Two cases of endothelioma of lymph-glands were wrongly interpreted, the first being mistaken for lymphadenoma, although the film was very atypical of this condition, and the second for chronic lymphadenitis.

A diagnosis of carcinoma of the vas deferens was made on one occasion in a case in which the testicle had been removed for gonococcal epididymitis. The inflammatory reaction in the walls of the vas was responsible for the unusual appearance of the epithelial cells.

Finally there were two cases in which the diagnosis given from the film cannot be explained and the mistake would probably be made again until further experience is available. The first film was made from a hæmangioma of the neck in which the cells seemed to show the features of a carotid body tumour, and the second from a ganglion of the palm which was undergoing organization, and this was thought to be a myxosarcoma.

In the whole series there were 17 occasions on which a section was not made from the material used for the wet film. These were mostly cases in which a 'rapid diagnosis' was given in the Out-patient Department or the operating-theatre, and since in this group there is definite evidence available that the diagnosis was correct, the numbers have been included.

Summarizing these results, it is evident that the number of cases in which an inaccurate diagnosis was given is extremely small, and the majority of these can be accounted for by errors in technique.

It remains to be said that there are many in which a more valuable and complete diagnosis was possible from the wet film than from the corresponding section. This occurred most frequently in the diagnosis of tuberculosis, which was often indefinite or doubtful in the sections and yet the picture was quite typical in the corresponding films. It is also a fact that in 8 cases a diagnosis of malignancy was made from the film, and although this was not demonstrated by the section

at the time, the fate of the patients confirmed the opinion. These cases are shown in *Table IV*.

*Table IV*

| Origin of Specimen | Wet-film Diagnosis | Section Diagnosis |
|---|---|---|
| Bone .. .. | Giant-cell sarcoma | Active new bone formation in and around fibrous myositis |
| Bladder .. | Carcinoma | No tumour present |
| Stomach .. | Carcinoma | No evidence of carcinoma |
| Tumour of leg | Spindle-cell sarcoma | Necrotic material |
| Uterus .. | Carcinoma | No evidence of malignancy |
| Thyroid .. | Carcinoma | Fœtal adenoma |
| Kidney .. | Carcinoma | Papilloma |
| Bladder .. | Carcinoma | Papilloma |

## SUMMARY

1. The method of examination of new growths and inflammatory diseases by wet films has been extended and applied to pathological processes in general.

2. Over 1000 cases have been investigated and the features of interest are reported upon and reviewed.

3. The application and technique of the method are described in detail.

4. The characteristic appearances of benign and malignant conditions are considered and the differential diagnosis is discussed.

5. The special histological features of various types of malignant cells are described and the suggestion that there is a definite difference between benign and malignant cells is put forward.

6. The finding of malignant cells in tissues which are apparently normal in appearance and at some distance from the original focus of disease has been possible in a large percentage of the cases examined with this object in view.

7. The possibility of early diagnosis of malignant change in benign growth such as papillomata of the bladder is recorded.

8. The errors in diagnosis which have been made are recorded and reference is made to the particular pitfalls which may be expected.

9. A number of instances are given in which the diagnosis suggested from the wet film was more complete or more accurate than that of the corresponding section.

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# RECONSTRUCTION OF THE FOREARM AFTER LOSS OF THE RADIUS

By R. WATSON JONES, Liverpool

The alluring prospect of healing by first intention in acute osteomyelitis provides a strong temptation to the surgeon to practise the operation of diaphysectomy. Unfortunately the early removal of the only available source of calcium and the collapse of the periosteal tube so commonly result in failure of regeneration that the operation cannot be performed with safety except in osteomyelitis of the fibula. In osteomyelitis of the radius, even with less destructive operations, there is a strong tendency to the development of radial club-hand with dislocation of the lower end of the ulna, and if the whole shaft of the bone is removed, the complication is almost inevitable (*Figs.* 7, 8).

The resulting deformity cannot be adequately treated by a bone-grafting operation. The difficulty lies not so much in the bridging of a wide gap filled with

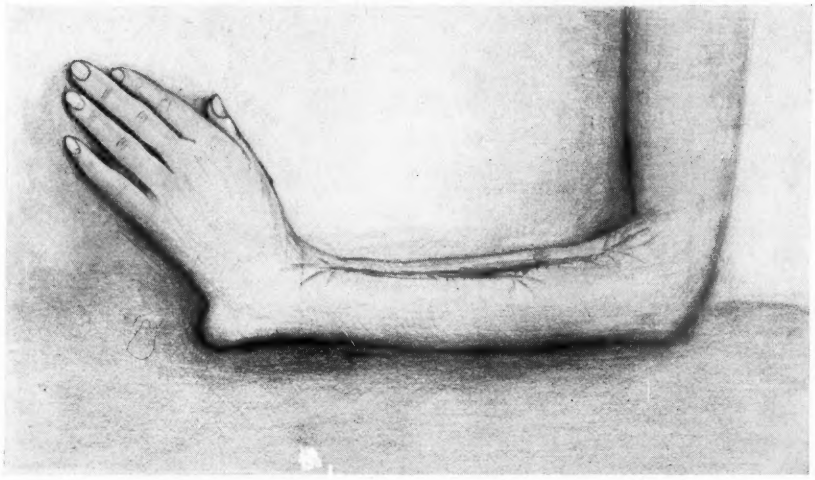

Fig. 7.—Failure of regeneration of the radius following diaphysectomy for osteomyelitis. Condition of the limb when first seen twelve months after original operation.

scar tissue between two very small fragments of bone, but in the preliminary replacement of the distal fragment of the radius in normal relationship to the ulna. If the inferior radio-ulnar dislocation is not reduced, the ugly prominence of the lower end of the ulna remains, and satisfactory radio-ulnar movement cannot be restored.

The operation described by Hey Groves[1] of implantation of the distal end of the ulna into the small distal fragment of the radius offers a solution to the problem, and this case is reported to illustrate the excellent cosmetic and functional result which may be anticipated from the construction of a single forearm bone.

The patient was a girl of 19 years, and diaphysectomy of the radius had been carried out by another surgeon twelve months before she was first seen. The old scar was excised, the lower end of the ulna dissected out subperiosteally, and angulated into a drill-hole in the fragment of the radius. Almost the whole length of the ulna was preserved in order to minimize the shortening of the limb, and the fragments were impacted in a position of 10° pronation from the mid radio-ulnar position (*Fig.* 9).

FIG. 8.—X-ray of forearm before reconstruction operation.

Although this construction of a single forearm bone involves complete loss of radio-ulnar movement, it is easy by internal rotation and slight abduction of the shoulder to bring the hand into the full palm-down position when resting on a table (*Fig.* 10), or by adducting and externally rotating the shoulder to turn the palm of the hand towards the face or the back of the neck (*Fig.* 11). Wrist movement has recovered to 80 per cent of normal, and it is not possible without a very careful examination to know that there is any abnormality of the limb.

FIG. 9.—X-ray of forearm two years after Hey Groves's operation (construction of a single forearm bone).

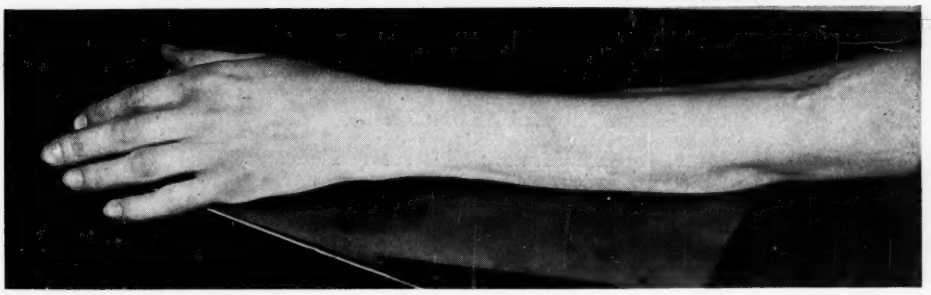

FIG. 10.—Photograph showing the excellence of the cosmetic result.

In 1932 Greenwood[2] described a modification of the operation; he divided the lower shaft of the ulna, overlapped the two ulnar fragments, and impacted the shaft into the small radial fragment, leaving the lower end of the ulna as a buttress "to give greater stability to the wrist-joint". Such a buttress develops, however, from subperiosteal bone formation even when the original technique is followed

FIG. 11.—Result of operation, showing that there is no functional disability.

(*see Fig.* 9), and the wrist-joint acquires normal stability. Greenwood's modification is unnecessarily complicated, it involves undue shortening of the limb, and leaves the disfiguring prominence of the lower end of the ulna, so that the cosmetic result is definitely inferior to that which may be secured from Hey Groves's original operation.

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# LOCALIZED HYPERTROPHIC ENTERITIS AS A CAUSE OF INTESTINAL OBSTRUCTION

## WITH A REPORT OF TWO CASES

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A REMARKABLE coincidence, with which most surgeons are familiar, is that the incidence of one rare and extraordinary case is so often followed closely by that of another of similar character. When the first of the two cases recorded below came under my notice I was so much puzzled to explain its nature that I was inclined to try to forget it; but when, within eighteen months, a second patient suffering from a precisely similar condition presented herself, I felt that a careful description and drawings should be prepared, so that others might have an opportunity of criticizing or explaining.

## CASE REPORTS

*Case* 1.—E. S., female, aged 37 years, married, was admitted to the Bristol Royal Infirmary on Aug. 1, 1931, and gave the following history.

HISTORY.—Twenty-four hours before admission she was suddenly seized with acute abdominal pain of a colicky nature, localized from its commencement to the right iliac fossa. This was followed by nausea and vomiting, and she had been unable to retain anything taken by the mouth. The bowels had not acted since the attack started, but she had suffered from some degree of constipation for years, which was easily relieved by mild aperients; otherwise she had been previously healthy and had not had any similar attacks of pain. The temperature was 99·6, the pulse 90, her tongue was dry and furred brown.

ON EXAMINATION.—The positive findings were tenderness, rigidity, and guarding in the right iliac fossa. The customary additional examinations yielded negative results and there was no palpable tumour.

The diagnosis of acute appendicitis, made by her own doctor, was confirmed, and operation immediately was advised and undertaken.

OPERATION.—The approach was made through a gridiron incision on the right side, and on digital exploration of the peritoneal cavity in the appendix area a section of bowel was felt which, to the touch, gave the impression of being an intussusception. The affected segment was easily identified as terminal ileum. The cæcum, appendix, and abnormal segment of bowel were delivered on to the abdominal wall. Although this delivery was effected with care and gentleness, it was apparent that the manipulation had resulted in perforation of the bowel wall, and it was this accident which determined the procedure eventually adopted.

The affected bowel, about 8 in. in length, was intensely congested, in parts almost black. It felt thickened, yet on handling proved to be fragile; there was no intussusception and no constriction ring. The commencement of both the congestion and thickening was sharply defined and terminated abruptly at the ileocæcal junction, the cæcum and appendix appearing normal. There was no evidence of thrombosis of the mesentery. On account of the diseased bowel having perforated, it was thought inadvisable to return this to the abdomen or to make any further encroachment upon the peritoneum, and a local excision was performed, the incision of the mesentery being made close to the bowel, in order to

preserve an efficient blood-supply to the remaining gut and to the anastomosis. The anastomosis was made between the end of the remaining ileum and the side of the cæcum, from which the ileocæcal valve had been removed, and was most difficult to accomplish. It was necessary to remove the appendix, as its mesentery prevented access to the cæcum.

A safety-valve drain, in the form of a catheter, was stitched into the anterior part of the junction line, and projected into the lumen of the ileum for about two inches, and finally the peritoneum around the aperture so made was stitched to the peritoneal edges of the incision. The remaining peritoneum, muscle layers, and skin were closed in the usual manner, the catheter being held in place by one silkworm-gut stitch to the skin edge.

SUBSEQUENT PROGRESS.—Three days after the operation the patient developed a sore throat which proved to be diphtheria, and, the surgical condition being considered satisfactory, she was removed by ambulance to the city isolation hospital. While there she developed paralytic complications, but in spite of this her surgical condition progressed favourably, and the fæcal fistula resulting from the drainage of the bowel closed rapidly after removal of the drain. The diphtheria and its complications terminated satisfactorily, and she was discharged, cured of all her ailments, some three months after admission.

PATHOLOGICAL REPORT.—The resected portion of bowel was examined in detail in the pathological laboratory attached to the hospital, and the report received reads as follows :—

The specimen is a portion of small bowel about 15 cm. in length. The wall is much thickened in most of its extent and measures 2 cm. at the thickest part, near the distal end, where the lumen is much narrowed. Near the proximal end the lumen becomes moderately dilated, but the bowel wall shows no thinning. The peritoneal surface is markedly congested, smooth, and glistening and shows no surface exudate. No thrombosed vessels can be seen.

The mucosa, over a very wide area, has been entirely replaced by a firm hæmorrhagic exudate, with roughened surface. At the proximal end the margin of this mucosal ulceration forms a clearly defined but irregular line. Although the healthy mucosa beyond shows some congestion, the transition from the diseased to the healthy segment is somewhat abrupt. Near the centre of the ulcerated mucosal surface and on the mesenteric side, the bowel wall is thinned out to a small ragged-edged opening, representing a more severely ulcerated area, which has been further damaged during the removal of this part of the bowel.

The microscopical examination reveals a condition of simple intestinal ulceration. The mucosa throughout the affected area has been completely destroyed and replaced by thick fibrino-purulent exudate. The only bacteria demonstrated in the ulcerated areas are those of the intestinal tract group, and of these streptococci are the predominating ones. There is no histological evidence of any of the granulomatous infections, and nothing in the nature of new growth can be found.

In the deeper layers and throughout the rest of the bowel wall no bacteria of any sort can be seen. In the bowel wall generally there is a marked acute inflammatory reaction, lessening in degree towards the subperitoneal layer, which shows congestion, œdema, and interstitial hæmorrhage but few polynuclear cells. This is in marked contrast to the condition in the submucosal and muscle layers, where, in places, the degree of polynuclear cell infiltration approaches abscess formation.

Evidence of previous acute inflammation of the bowel is present in the form of much dense scar tissue. There is much fibrotic thickening of the submucosa, and extensive areas of the muscle coat have been entirely replaced by fibrous tissue. Beyond a very occasional small vessel there is no evidence of vascular thrombosis.

The accompanying excised appendix is normal, and on section shows no evidence of inflammatory change.

*Case* 2.—I. T., female, aged 25 years, unmarried. Admitted to the Bristol Royal Infirmary on Jan. 1, 1933, complaining of acute abdominal pain which had commenced forty-eight hours before admission and was localized to the right iliac fossa during the whole of this period. Vomiting was a prominent symptom, and she had not been able to retain anything taken by mouth.

HISTORY.—The patient's previous history revealed the fact that since childhood she had suffered from mild dyspepsia, not constant or sufficiently severe or continuous to demand medical attention, and never accompanied by vomiting; and that she had always had severe dysmenorrhœa of the congestive type. She was not menstruating at the time

of admission. The bowel action had always been regular, with a slight tendency to diarrhœa, which she never regarded as of any importance, so little inconvenience did it cause. She had never had an attack of pain similar to that from which she was suffering on admission.

ON EXAMINATION.—The temperature was 100° and the pulse 96, the tongue furred, brown, and dry. The positive findings on examination were similar to those in *Case* 1 and no tumour was felt in the abdomen. It was thought that she was suffering from acute appendicitis, the diagnosis made by her own doctor, and urgent operation was advised, to which she consented.

OPERATION.—The same method of approach was used as in the operation just described; the results of the local exploration and delivery of the diseased bowel exactly correspond, even to the perforation of the gut on gentle manipulation. The appearance of the lesion, its distribution, consistency, and colour left no doubt that the condition was the same as that encountered in the previous case, and again the extent of the operation was limited by a perforation. The difficulty of performing efficiently an anastomosis by the method adopted before led to a modification in the technique, which was as follows: The diseased segment of gut was excised and the terminal end of the proximal ileum closed in the manner ordinarily adopted in intestinal resection, as also was the cæcum at the site of the excised ileocæcal valve. A side-to-side anastomosis was carried out, ileum to anterior cæcal wall. The gut was not drained. This modification rendered the operation easy, and would be adopted by the writer without hesitation if a similar condition should ever be encountered again.

SUBSEQUENT PROGRESS.—The patient made an uninterrupted recovery, except that at one small area in the scar some hypertrophic granulation tissue, which did not yield to the application of caustics, required to be cut away. This area corresponded to the site of a small rubber dam drain which was left in the incision down to peritoneum for the first three days after operation. Although convalescence was uncomplicated, it was slow, and the patient was not able to return to her work, domestic employment, until five months after the operation. She is now well, but volunteers the statement of two interesting facts: (1) That whereas before the operation her bowels were inclined to be loose, she now has to take laxatives occasionally or she becomes constipated; and (2) That her dysmenorrhœa is absolutely cured.

PATHOLOGICAL REPORT.—The pathologist's report on the specimen removed is almost word for word that given on *Case* 1, with the minor exceptions that the length of the excised bowel in this case is 20 cm. and the bacteria present do not include streptococci and are comparatively scanty.

The illustrations are taken from this case; *Fig.* 12 represents the appearance of the diseased bowel at the time of operation. *Figs.* 13, 14 are copied from the actual specimen, and the microphotographs (*Figs.* 15, 16) are also taken from sections cut from the same preparation.

## COMMENTARY

The writer has not been able to discover in the surgical literature available an account of any parallel case, and the diseases to which the ileum is liable do not correspond in any instance with the condition found in these patients.

Apart from the interest aroused by the discovery of so massive a lesion with so short a history, at any rate of severe symptoms, the questions of nomenclature, diagnosis, and treatment arise. It is a difficult task to name appropriately a lesion which shows such a variety of diverse pathological changes without any obvious causative factor. At first sight the appearance of deep congestion in the gut and its sharply limited extent strongly suggest that there has been some strangulation. An internal hernia, strangulation by a band, or a volvulus might each cause this appearance of abruptly limited strangulation, or a thrombosis of one of the terminal branches of the ileocolic artery might have produced the same apparent result. But even if we ignore the fact that neither incarceration, twisting, thrombosis, nor strangulation by band were found, we are confronted by two facts which prove that the causative condition must have been of a somewhat chronic nature. The

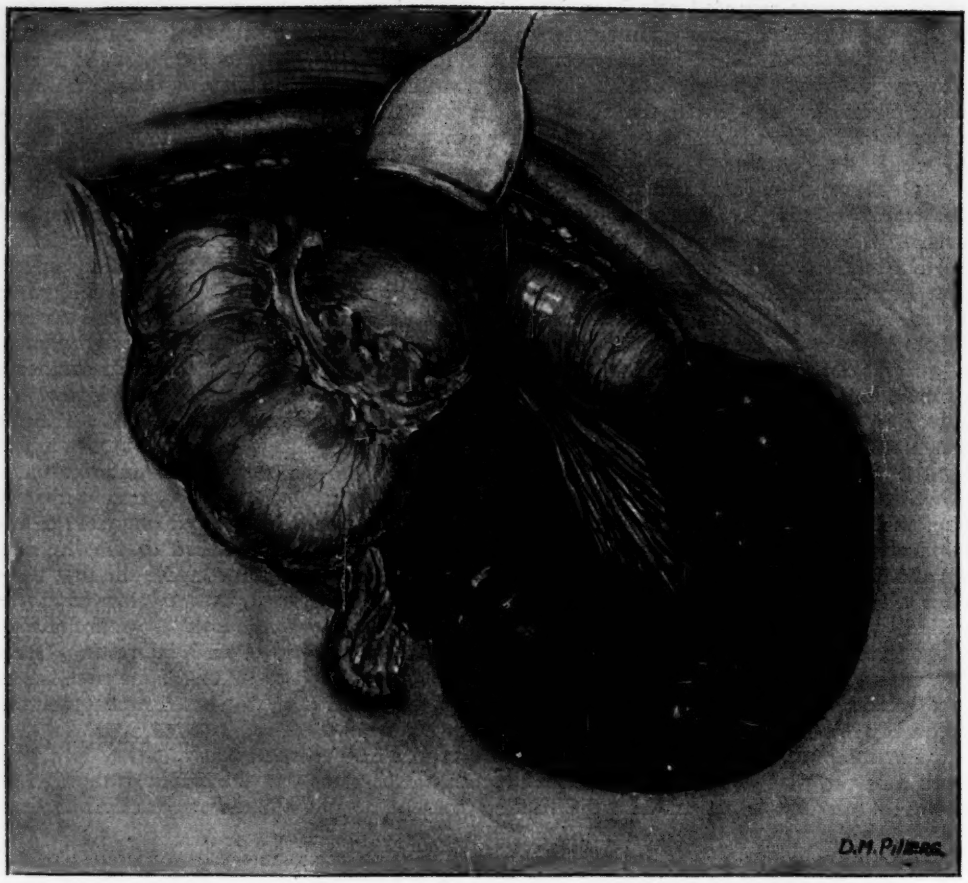


FIG. 12.—Condition of ileum found at operation. Note abrupt commencement and termination of inflammatory lesion.

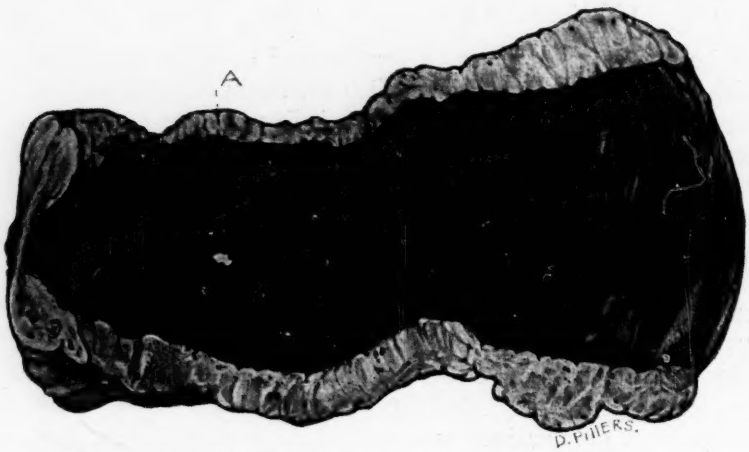


FIG. 13.—Portion of ileum removed, opened by longitudinal incision, showing the intense congestion of the mucous surface, extreme fibrous thickening of the intestinal wall. Note extensive necrosis of small area and perforation (A).

FIG. 14.—Transverse section to show thickened wall of ileum and obliteration of lumen.

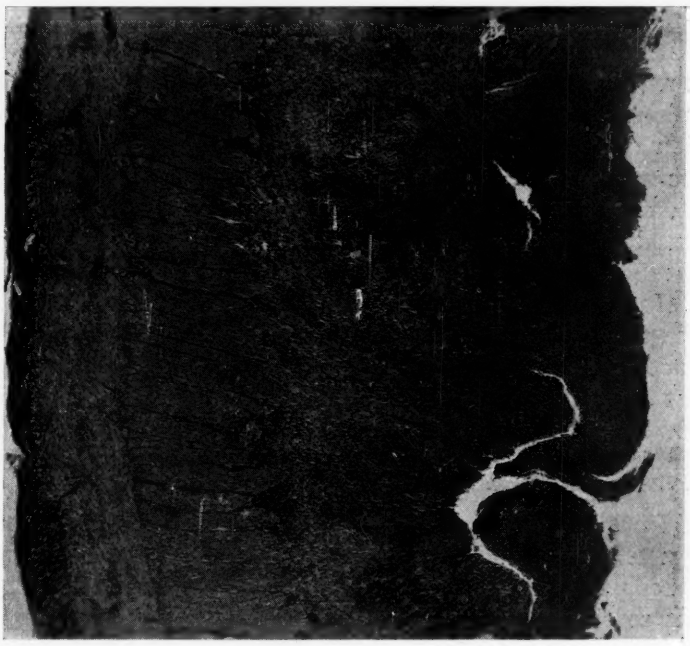

FIG. 15.—Section near the proximal end of the diseased portion of bowel to show the ulcerated mucosa and the intense inflammatory reaction in the submucosa. Shows also extreme thickening, but for technical reasons it was not possible to obtain a 'full thickness' photograph at the most thickened portion. (× 20.)

FIG. 16.—Section through the junction between diseased and healthy bowel. The space is due to contraction of the tissues during preparation of the section. (× 90.)

first of these is that there is enormous thickening of the bowel wall, consisting of well-formed fibrous tissue, and the second that the mucous membrane has been almost completely destroyed by ulceration. The main change seems to be that of hypertrophic reaction to an inflammatory process which is mainly in excess of the destructive ulceration present. 'Subacute hypertrophic ulcerative enteritis' is the designation suggested.

It will be noticed that the symptoms and signs are not quite typical of acute appendicitis, and to cite one point which did suggest some other condition, the pain commenced in and remained in the right iliac fossa. There are many who will agree that this fact alone should always demand an exhaustive search for some other lesion. In the present instance one might have expected the discovery of a sausage-shaped tumour on palpation of the abdomen, a sign which was not elicited, probably on account of the rigidity of the abdominal muscles on the affected side.

The most common positions in which the appendix is situated are the retro-cæcal sites and the pelvic situation, the splenic, inguinal, and promonteric positions being comparatively rare. When inflamed and in the first-mentioned position loin tenderness can almost always be elicited, and in the pelvic site tenderness in the pouch of Douglas. These signs were absent in the present instances.

It is suggested that the diagnosis of the disease described here might be borne in mind in cases presenting atypical features in history or signs of acute appendicitis, and that considerable effort should be directed to the finding of a tumour which should be palpable.

In regard to the treatment it may be as well to consider alternatives first. The diseased gut might have been left *in situ* in the hope that it would undergo spontaneous cure. This seemed, and still seems, to be taking a graver risk than that of active intervention; the issue is impossible to determine without the benefit of the experience of others who, faced with like circumstances, have adopted the conservative course.

Then again, a more radical and orthodox operation might have been performed, which would actually have been easier and would have assured vascular stability in accordance with recognized anatomical considerations.

In defence of the procedure adopted it can be said that the blood-supply to the remaining bowel was never in any doubt. Before the anastomosis was commenced the ileum and cæcum were watched for several minutes, in order to determine the fact that they possessed a blood-supply sufficiently efficient to promote the necessary healing of the anastomotic line.

The object of all treatment, medical or surgical, is to restore our patients to their normal physiological balance and, as far as possible, to preserve their designed anatomical conformation with the object of the restoration of normal health. This object has apparently been attained by the method defined here.

I am indebted to Dr. A. D. Fraser, who has so carefully prepared the pathological reports and the microphotographs and has taken infinite pains to elucidate the lesion and to discover a similar condition in the literature of pathology. My thanks are due also to Professor E. W. Hey Groves for his interest and help in many ways, to Mr. C. F. Walters and Mr. A. W. Adams at whose request the cases came under my care, and to Miss D. M. Pillers, who has so faithfully reproduced in the sketches the features which I desired to demonstrate.

# GASTROJEJUNAL ULCERATION

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Our experience of the nature and frequency of gastrojejunal ulceration is of necessity short, for the first gastro-enterostomy was performed by Wolfler, at the suggestion of his assistant Nicoladoni, as recently as the year 1881. Possibly because the earlier operations were generally performed for carcinoma of the pylorus, the first case of gastrojejunal ulcer was not reported by Braun[9] until 1899, this being one in which a fatal perforation took place. In the short period of thirty-five years that has ensued since this date our knowledge has been steadily increasing. At first only single cases were reported at long intervals, the first in France being reported by Quénu in 1902, and the first in this country by Mayo Robson in 1904. After this they were found in rapidly increasing numbers, Paterson,[39] in his epic paper in 1909, first directing widespread attention to the subject, although he found that Brodnitz[19] in 1903 was able to collect 15 cases, Gossett[17] in 1906 31 cases, and Einer Kay[13] a year later added 6 examples. Paterson in his own paper collected 52 certain and 11 doubtful cases, 2 of which were from his own practice.

Of later years so many examples have been reported that it is no longer practical to collect individual cases but only to consider general statistics. So many indeed have been reported that a keen controversy has arisen as to the actual frequency of the condition. On the one hand, Lewisohn[28] in 1925 published a series of 68 cases of gastro-enterostomy where 23, or 34 per cent, developed gastrojejunal ulceration after having been watched for a period of not less than five years. Hurst and Stewart[24] believe that it is a dangerous and frequent sequel of gastro-enterostomy. They cast doubt upon the accuracy of many of the figures reported by various surgeons, and claim that the number of anastomotic ulcers slowly but steadily multiplies as the cases of gastro-enterostomy are watched over increasing periods. They give pathological statistics showing a 7·3 per cent incidence in 42 cases examined at Leeds, ten days to two months after operation, and a 52 per cent incidence in 42 cases examined nine months to nineteen years after operation. These, however, must be considered as very selected cases, for the examinations were carried out on patients who had died, and therefore took no cognisance of those who had recovered and might have formed a high percentage of the total operated upon. These extreme views as to the frequency of this dangerous complication are held by a considerable number of surgeons of wide experience. This is especially so on the European continent, where many have abandoned the routine use of gastro-enterostomy and have replaced it by some form of partial gastrectomy. Among those who hold these views may be quoted Finsterer,[14] von Haberer,[47] Hohlbaum,[22] von Eiselsberg,[44] and in this country Pannett.[38]

On the other hand, many surgeons, and especially those in this country and the United States, have found that recurring ulceration of this type, although

dangerous and not entirely unavoidable, occurs in only a small percentage of the cases, and is insufficiently frequent to warrant the abandonment of so useful an operation as gastro-enterostomy. Paterson in 1909 found only 3 gastrojejunal ulcers in his own series of 348 cases, and in a later paper found 12 examples in 495 cases—that is, 2·4 per cent. Lord Moynihan[37] in 1919 found 10 cases in 613 gastro-enterostomies, or 1·6 per cent. Balfour[2] found that a recurrent ulcer developed in only about 3 per cent of the gastro-enterostomies performed for duodenal ulcer. In a series I published in 1925[48] there were 631 cases of gastro-enterostomy with 11 gastrojejunal ulcers—that is, 1·7 per cent. Of these, 417 were performed for pyloric and duodenal ulcers with 10 recurrent ulcers—that is, 2·4 per cent. In a later series published in 1930[49] there were 616 gastro-enterostomies, 16 subsequently developing gastrojejunal ulcers—that is, 2·6 per cent. These figures agree very closely with those published by Luff[30] from the collective investigation of the British Medical Association, where, of 995 cases of duodenal ulcer, 744 were traced; of these 21 (2·8 per cent) had a subsequent history of gastrojejunal ulcer.

Many of the published figures are, however, depreciated in value by the fact that no clear distinction is made in the statistics between cases operated upon for gastric or duodenal ulcer. Moreover, the criticism is not infrequently made that the cases have not been sufficiently carefully followed or have been watched for too short a time. In this respect it may be important to remember that physicians are likely to see only the surgical failures, just as the surgeons are only asked to treat patients resistant to medical treatment, and thus each is prone to believe that there is a much higher percentage of such failures than really exists.

It was in the belief that light might be thrown upon some of these much discussed points that the Association of Surgeons undertook a collective investigation into the subject of gastrojejunal ulcer. The subject matter of the present communication is based upon a study of my own cases prepared for this investigation.

## TOTAL INCIDENCE

By the courtesy of the London Hospital authorities I have been able to establish a Follow-up Department which has been in existence since 1919. In this department all cases are examined at intervals which at first are quite short and later are increased as the patient remains well. This has been so successful that I feel confident that no case of gastrojejunal ulcer has escaped my notice; although several have been treated and operated upon elsewhere, a record has always been procured. I can therefore feel that the figures quoted are reliable and are as accurate as can be obtained for any series of cases under the care of one surgeon.

For the collective investigation it was decided to consider only the cases which were operated upon in the years 1926–30 inclusive. The frequency of gastrojejunal ulceration in this group is shown in *Table I*, and it is seen that the incidence of this complication was 0·73 per cent. It was realized, however, that these figures are considerably below those which I had previously published, and it was thought that this might be due to the fact that the series contained too few cases and that a more correct estimate might be obtained by a study of all the cases which had occurred in my practice. These figures are shown in *Table II*, and it will be seen that, excluding 18 cases of gastrojejunal ulceration where recurrence followed a local operation, there were 31 gastrojejunal ulcers following 1859 gastric operations

—that is, an incidence of 1·6 per cent. One of these cases was a recurrence after a partial gastrectomy for a gastrojejunal ulcer, so that there were really only 30 cases of fresh marginal ulceration.

*Table I.*—Cases Operated upon 1926–30

| | Total | M. | F. | Traced | Gastro-jejunal Ulcer | Per Cent |
|---|---|---|---|---|---|---|
| Duodenal ulcer .. | 319 | 252 | 67 | All | 4 | 1·22 |
| Gastric ulcer .. | 254 | 167 | 87 | All | — | — |
| Gastric carcinoma .. | 71 | 44 | 27 | All | — | — |
| Gastrojejunal ulcer | 32 | 28 | 4 | All | 1* | 3·10 |
| Totals .. | 676 | 491 | 185 | All | 5 | 0·73 |

* Recurrent.

*Table II.*—All Cases

| | Total | M. | F. | Traced | Gastro-jejunal Ulcer | Per Cent |
|---|---|---|---|---|---|---|
| Duodenal ulcer .. | 893 | 737 | 156 | All | 29 | 3·24 |
| Gastric ulcer .. | 683 | 446 | 237 | All | 1 | 0·14 |
| Gastric carcinoma .. | 221 | 148 | 73 | All | — | — |
| Gastrojejunal ulcer | 62 | 58 | 4 | All | 1* | 1·60 |
| Totals .. | 1859 | 1389 | 470 | All | 31 | 1·60 |

| | Total | M. | F. | Traced | Ulcer |
|---|---|---|---|---|---|
| Gastrojejunal ulcer (other operations) | 18 | 15 | 3 | All | 6* |

* Recurrent.

Those who believe with Hurst[24] that the cases increase in number the longer the patients are followed will regard this increased incidence as due to the inclusion in the list of many cases operated upon before 1926. This is in part true, but it is not the sole explanation. My own cases date back to 1913. In 1923 the statistics were carefully investigated and published, and the present series was prepared to include cases up to September, 1933, so that the two ten-year periods can be easily compared. In the ten years, 1913–22 inclusive, there were 765 gastric operations, and the present investigation shows that of the 30 patients who had gastrojejunal ulcers 18 were operated upon in this period, giving an incidence of 2·3 per cent as compared with the total of 1·6 per cent.

## THE NATURE OF THE PRIMARY LESION

It is now a well-recognized fact that this complication is more prone to follow operation for certain types of ulcer than for others. Paterson[39] had noted this in his paper in 1909, when he stated that 33 of the certain 52 cases followed operation

performed for pyloric stenosis, and found only 1 case, that reported by Einer Kay, following gastro-enterostomy performed for gastric carcinoma. Hurst[24] quotes Judd[26] as having likewise known, up to 1921, only 1 case occurring after operation for gastric carcinoma. He also states that it occurs but rarely in cases of gastric ulcer, but is a frequent sequel after duodenal ulcers. These statements are well supported by the present series (*Tables I* and *II*), for it will be seen in *Table II* that, whereas 29 ulcers occurred after 893 operations for duodenal ulcer (3·24 per cent), there was only one after 683 operations for gastric ulcer (0·14 per cent), and not a single example after 221 operations for gastric carcinoma. The highest incidence therefore is among duodenal ulcers. The figures for the cases operated upon in 1913–22 inclusive give 457 gastro-enterostomies for duodenal and pyloric ulcers, with 18 gastrojejunal ulcers, a maximum incidence of 3·9 per cent, all of these cases having been followed for a period of over ten years.

The actual number of cases of gastrojejunal ulcer included in this investigation is 79, for in addition to the 30 cases occurring in those patients where I had personally operated there were 49 in whom the primary operation was performed elsewhere. In these 49 cases there was only 1 in which it could not be certain that the first operation was performed for a pyloric or duodenal ulcer. It may be accepted, therefore, that gastrojejunal ulceration is a complication almost entirely limited to gastro-enterostomy performed for ulcers in the neighbourhood of the pylorus or duodenum. After operations for gastric ulcer or carcinoma the condition is so rare as to be negligible.

## THE VARIETY OF PRIMARY OPERATION

Although marginal ulceration is more common after operations for duodenal ulcer than for other lesions, there is evidence that the incidence varies according to the nature of the anastomosis that has been performed. Even in his early paper Paterson stated that it was believed to be more common after anterior than posterior gastro-enterostomy, for there were 27 anterior anastomoses, 10 anterior with a lateral anastomosis, 2 'in Y', 12 posterior, and 1 supracolic. He believed that the apparently greater frequency after the anterior operation was due to the fact that with it a lateral anastomosis was more often performed, and therefore, as also after the 'in Y' operation, the acid was not neutralized.

Lord Moynihan[37] in 1919 found 6 examples after 189 anterior, and 4 after 444, posterior anastomoses. Wright also believes that the lesion is more common after the anterior anastomosis, for he found in his series that the anterior operation had been performed in 72 instances and the posterior in 59.

That the incidence is higher after the performance of a lateral or an 'in Y' anastomosis is almost certain, as will be shown later when the relation of such ulceration to hyperacidity is considered, but it is difficult to believe that it is more common after a simple anterior anastomosis. In my own series this has certainly not been the case (*Tables III* and *IV*). In *Table III* the cases operated upon in 1926–30 inclusive are alone considered, and whereas after 446 posterior anastomoses there were 4 gastrojejunal ulcers—that is, 0·89 per cent—there was not a single example after 33 anterior unions. In *Table IV* all cases are considered, and 1313 posterior anastomoses were followed by 29 gastrojejunal ulcers—that is, 2·2 per cent—but 77 anterior unions again showed no cases of marginal ulceration. In the 49 cases

where the operation had been performed elsewhere there were 43 examples following the posterior and 4 following the anterior union (*Table V*). In two of the latter a lateral anastomosis had been performed.

*Table III.*—Cases Operated upon 1926–30

| | Total | Traced | Gastro-jejunal Ulcer | Per Cent |
|---|---|---|---|---|
| Posterior gastro-enterostomy .. | 446 | All | 4 | 0·89 |
| Anterior gastro-enterostomy .. | 33 | All | — | — |
| Anterior Polya .. .. | 16 | All | — | — |
| Posterior Polya .. .. | 181 | All | 1 | 0·55 |
| Totals .. | 676 | All | 5 | 0·73 |

It is widely believed that the lesion does not occur after the operation of partial gastrectomy, and it is acting on this belief that certain surgeons, as already mentioned, have replaced gastro-enterostomy by some form of partial gastrectomy in the treatment of duodenal ulceration. A considerable number of such recurring ulcers have, however, now been described. Hoguet and Cole[21] reported the presence of a large marginal ulcer occurring five years after the performance of the Polya operation for a duodenal ulcer. Beer[4] and Wright[54] also report cases, and the latter suggests that the popularly accepted belief that these ulcers are rare after partial gastrectomy

*Table IV.*—All Cases

| | Total | Traced | Gastro-jejunal Ulcer | Per Cent |
|---|---|---|---|---|
| Posterior gastro-enterostomy .. | 1313 | All | 29 | 2·20 |
| Anterior gastro-enterostomy .. | 77 | — | — | — |
| Partial gastrectomy .. .. | 469 | — | 2* | 0·43 |
| | 1859 | — | 31 | 1·60 |
| Other operations .. .. | 313 | — | 6† | |
| Totals .. | 2172 | — | 37 | 1·70 |

* One recurrent. † Recurrent.

is probably due to the fact that this operation is most frequently performed for carcinoma of the stomach, following which a gastrojejunal ulcer is rare. Hurst[24] quoted Birgfeld[5] as having collected 67 cases from the German literature, and states that he has himself seen reports of 14 cases published since then. Three cases had occurred at the New Lodge Clinic, 2 of which were performed for gastrojejunal ulcer. Haberer[46] reported a case occurring seven years after the performance of a Billroth II operation for duodenal ulcer, and for this reason returned to a modification of the Billroth I operation, where he closed the stump of the duodenum and implanted the cut end of the stomach into the anterior surface of the duodenum below the closed opening.

In my own series (*Table IV*) there were 469 partial gastrectomies, in 2 of which gastrojejunal ulcers followed—that is, 0·43 per cent : 143 of these operations, however, were performed for carcinoma, so that of 326 operations performed for ulcers 2 examples of marginal ulceration were seen—that is, 0·61 per cent. Of these two cases, one followed a Polya gastrectomy for a marginal ulcer occurring after a posterior gastro-enterostomy performed elsewhere, and was definitely due to a technical error, in that, owing to extensive adhesions, I was content to remove a smaller portion of the stomach than usual. A second more extensive operation was performed, and the patient has since remained well. The other was an acute ulcer occurring after a Polya resection, and as the operation was performed in 1915, a smaller portion of the stomach was removed than would have been taken to-day. In the 49 cases of marginal ulcer in my series in which the primary operation had been performed elsewhere (*Table V*), there were also 2 examples following partial gastrectomy, in both cases of the Billroth II variety.

*Table V.*—GASTROJEJUNAL ULCERS

| | TOTAL | M. | F. | DUODENAL | LESSER CURVE | P.G.E. | A.G.E. | BUTTON | 'IN Y' | P.G. | SILK IN ULCER |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Self .. | 30 | 29 | 1 | 29 | 1 | 29 | — | — | — | 2† | 2 |
| Others | 49* | 43 | 6 | 48 | 1 | 43 | 4 | 1 | 1 | 2 | 13 |

* In this group there were 51 operations in 49 cases, for two patients had recurrent ulcers.
† One recurrent.

There seems therefore no doubt that a partial gastrectomy does not give a complete immunity to the onset of gastrojejunal ulcer ; but it is less frequent after this operation—about 0·5 per cent—than after gastro-enterostomy. My own belief is that, provided sufficient of the stomach is removed to produce an achlorhydria, the complication is almost unknown, although the risk of subsequent anæmia may be increased thereby.

From time to time much stress has been laid upon the fact that gastrojejunal ulcer does not follow gastroduodenostomy. This must of course be so. If there is no gastrojejunal anastomosis there can be no gastrojejunal ulceration. Its place would be taken by recurrent ulceration. A full discussion of the question of such recurrence is outside the scope of this paper, but it may be stated that Grey Turner,[18] in a series of 43 such cases, found 4 recurrences.

## THE RELATION OF MARGINAL ULCERATION TO OPERATIVE TECHNIQUE

It has frequently been suggested that the incidence of marginal ulceration is increased by technical errors in the operation. In the earlier cases it would seem probable that the use of mechanical devices, such as a Murphy's button or a bobbin, by causing necrosis of the opposed edges, instituted a process of ulceration. This might have progressed to a chronic marginal ulcer, and the inclusion of a large number of such cases might account for the exceptionally high incidence of this

lesion in some of the earlier series that have been reported. Garnett Wright[54] states that in his series of 155 collected cases a Murphy's button or some form of bobbin had been used in 14. In my own series there was no example in the 30 cases following my own primary operation, for I have never used any method other than a simple suture, but in the 49 cases following operation performed elsewhere there was one in which a Murphy's button had been used for the anastomosis after a perforated duodenal ulcer. The button was still in place and was associated with a chronic marginal ulcer.

At one time many surgeons, including myself, were of the opinion that the use of unabsorbent sutures was an important factor; however, the knowledge that such ulceration occurs when catgut has alone been used makes this position no longer tenable, but the presence of pieces of silk in the base of a large number of these ulcers would lead to the belief that its use might increase the incidence. Garnett Wright,[54] in his series of 155 cases, found 13 in which portions of such suture could be found. Many other similar cases have been reported, but Hurst[24] believes that the importance of this factor has been exaggerated, as pieces of suture may be present without ulceration. In my own 79 cases there were 15 examples, in 2 of which I had performed the primary operation myself. In some of these the suture arose from the centre of the ulcerated area and it was difficult not to conclude that it was a factor of some importance (*see Fig.* 19). But since in 64 cases no suture could be found even by microscopical examination, it is evident that this is not so important a factor as was at one time believed. The use of catgut may reduce the incidence but it will not abolish it.

It has been claimed that the crushing effect of clamps applied during the process of anastomosis might initiate the ulcer, which would slowly progress. The main answer to this contention is that, as with ulcers of the stomach and duodenum, it is now known to be a very easy matter to produce acute ulceration experimentally, but unless some other factor is present, such ulcers always heal rapidly and cannot be induced to progress to a chronic state. If the other factor is present, ulceration will occur with or without the presence of primary trauma. Bolton[7] states that an acute toxic ulcer produced in an animal invariably heals soundly in three to four weeks. Paterson[40] states that there are two main reasons against the acceptance of this view—firstly, that there is too long an interval between the operation and the onset of symptoms of marginal ulceration, and, secondly, that the obstruction does not as a rule occur at the site where the intestine is gripped by the clamps. In the 79 cases in this series there was not one in which the ulcer was so situated as to give rise to the belief that it could have originated in this way. Gronnerud[20] carried out a series of experiments in dogs showing that the incidence of this complication was unaffected by the use of clamps, and in many of the reported cases in man no clamps have been used. It may therefore be accepted that if clamps are employed with ordinary care they are not a factor in the production of this lesion.

It would seem reasonable to believe that if care were not used to obtain accurate apposition of the mucosa a raw area might be left at the line of suture which, after being acted upon by the acid juice, would progress to a chronic peptic ulcer. Grey Turner[19] has laid stress upon this possibility and shown the importance of obtaining accurate mucosal apposition. My attention was first directed to this possibility by the fact that several of the ulcers in those cases where I performed the first operation

myself were placed in a different situation from those seen when the first operation had been performed elsewhere. The majority of surgeons terminate their anterior line of through-and-through sutures at one or other extremity of the mucosal incision and it was in this position the new ulcer was found (*see Fig.* 17). It has been my own custom to terminate this suture about the middle of the incision so that I turn the corners with a continuous suture. In several of my cases the ulcer was situated at this point (*see Fig.* 18). In either case the ulcer was placed at the last half inch of the through-and-through suture line where the mucosa is most likely to slip back and thus escape inclusion in the union. For this reason I have for several years past abandoned excision of the redundant mucosa. The line of suture is less neat in appearance, but one has the feeling of certainty that there is complete mucosal apposition throughout the whole length. Even with such precautions ulceration still takes place. If the formation of a raw area is a factor, it is a small one.

At one time great stress was laid upon the effect of pyloric occlusion in increasing the incidence of gastrojejunal ulceration. Martin[33] supports this view, for he states that the pancreatic secretion is normally activated by the passage of acid through the pylorus. The contact of this acid with the duodenal mucosa produces secretin, which is carried by the blood-stream to the pancreas, where it stimulates the secretion of pancreatic juice. If the pylorus is occluded, this mechanism is disturbed. Galpern[16] stresses the importance of this fact in surgery and shows that if a dog's stomach is divided into two halves, the introduction of acid into the cardiac end has no effect, but if it is inserted into the pyloric portion a flow of pancreatic juice immediately follows. Von Haberer[47] supported this view on his clinical experience, for he found that after 275 cases of simple gastro-enterostomy there were 3 examples of jejunal ulcers, and after 71 cases with pyloric occlusion there were 12 jejunal ulcers. Paterson[41] is, however, entirely opposed to this view, for he has found that after gastro-enterostomy there is a diminution of the total chlorides as well as of the free HCl in the gastric juice. He regards this as being due to the fact that the hormone regulating the flow of HCl is formed in the pyloric end of the stomach and is not utilized if the food does not enter this portion. Whatever is the effect of attempted permanent occlusion is of minor importance, however, as this step is rarely practised to-day. Temporary occlusion with a silk stitch would seem to have very little bearing. I invariably practice this step, but the incidence of marginal ulceration in my own cases has been no higher than in series reported elsewhere.

As a result of this tedious but necessary discussion it would appear that the following conclusions can be accepted as accurate: (1) Marginal ulceration is almost limited to cases of anastomosis for duodenal ulcer; (2) After all gastric operations its incidence is 1·6 per cent; (3) After all gastric operations watched for ten years its incidence is 2·3 per cent; (4) After all posterior gastro-enterostomies its incidence is 2·2 per cent; (5) After all posterior gastro-enterostomies for duodenal ulcer its incidence is 3·24 per cent; (6) After all posterior gastro-enterostomies for duodenal ulcer watched for ten years its incidence is 3·9 per cent (this must be considered as the highest possible incidence); (7) The condition is less common after partial gastrectomy, the incidence being only 0·61 per cent; (8) Technical errors in the primary operation may increase the frequency of the lesion, but are not the primary cause.

## THE CHOICE OF OPERATION

Since after partial gastrectomy there is a much lower incidence of marginal ulceration, it would seem evident at first sight that it should be the operation of choice, but it is necessary to be certain before a decision is made that this more extensive operation is not associated with a higher mortality. In investigating such a question there are always great difficulties, for the operative mortality is the one fact which alters with the experience of the surgeon and therefore is to a large extent personal. Much time is wasted in surgical meetings discussing the best operation for a given lesion. Preferably the question should be: Which is the best operation in the hands of a given surgeon? One surgeon may perfect one method and another a second. Nevertheless these operations have now been so widely performed that a fair estimate can be made of the operative mortality with either method obtained by the more experienced surgeons.

Luff,[30] in his collective investigation, gave the total mortality for posterior gastro-enterostomy as 5 per cent, and Conybeare[12] states that of 190 cases admitted to Guy's Hospital the mortality was over 5 per cent. Individual series show, however, much better results. Mayo[35] obtained in 4532 cases of duodenal ulcer a mortality of 1·76 per cent. Balfour[3] found in 492 cases 9 deaths—that is, 1·82 per cent—but in his report for 1930[2] there were 524 cases with only 6 deaths—that is, 1·14 per cent. Lord Moynihan's series of 1000 cases without a death has frequently been commented on. These are, however, the statistics of exceptionally skilled surgeons, and can be equalled by but few. In this country, indeed, there is a tendency for the mortality of gastro-enterostomy in the hands of specialists to increase, for the simple cases are now performed locally and only the patients who —owing to old age, concomitant disease, past severe hæmorrhage, or extreme stoutness—must be considered as bad surgical risks are sent to the specialists. In my own series analysed up to November, 1929, there were 550 cases with 7 deaths—that is, a mortality of 1·2 per cent—and this included a consecutive series of 263 cases without a death. In the series analysed up to August, 1933, the total mortality was 2 per cent, and it is probable that this figure should be regarded as the average mortality for this operation. Although perhaps not germane to the present discussion, it must also be remembered that many surgeons have been able to publish series of gastro-enterostomies giving 80 to 90 per cent of cures. I have published[52] a series of my own cases where 88 per cent were cured after being followed for a minimum of five years.

The mortality of partial gastrectomy performed for duodenal ulcer is also difficult to estimate, for many of the reported series of partial gastrectomies include cases of ulcer of the body of the stomach, so that the two series are not comparable. Probably the most satisfactory figures that have yet been published are those of Finsterer,[14] who in a total of 566 cases had only 18 deaths, a mortality of 3·1 per cent. But with the Billroth I method it was 5·8 per cent. Burke[11] gives the results of cases operated upon at the Eiselsberg Clinic in Vienna; there were 606 cases with a mortality of 5·44 per cent. Louria[29] reports the results of cases operated upon by von Haberer at the University Clinic at Graz; there were 284 cases with 24 deaths, a mortality of 8·4 per cent. Schoemaker[43] reports results of 350 cases operated upon by his method; there were 17 deaths—that is, 5 per cent. Balfour in 1929 published a series with a mortality of 5·5 per cent, and the collected statistics

of Luff[30] gave a 6 per cent mortality. In my own series a partial gastrectomy was so seldom performed for a duodenal ulcer that I have no comparable statistics.

From the above figures it would seem fair to estimate that the mortality after gastro-enterostomy for duodenal ulcer is in the neighbourhood of 2 per cent and after partial gastrectomy about 6 per cent. That is, the mortality of the latter operation is slightly higher than the combined mortality and the incidence of recurring ulcer after posterior gastro-enterostomy, and this does not take into consideration the incidence of recurring ulcer after partial gastrectomy.

Since, as will be shown later, at least 75 per cent of gastrojejunal ulcers can be cured by a subsequent partial gastrectomy it would seem manifest that the most satisfactory operation for duodenal ulceration is posterior gastro-enterostomy, a partial gastrectomy being reserved for the small percentage of cases which later develop gastrojejunal ulceration.

## PATHOLOGY

The pathological characters of these ulcers are identical with peptic ulcers seen in the stomach or duodenum. A few cases of acute ulceration are seen; these are either operated upon for severe hæmatemesis or are discovered post mortem after a perforation or death from some other cause; they are therefore usually found shortly after operation. In 3 of the 79 examples in my series there were intervals of one month, one month, and five months respectively after the first operation. In such cases there are one or more areas of sharply cut but irregular ulceration involving only the mucosa but tending to encircle the anastomotic area. The edge of the mucosa may be free or only lightly attached to the underlying tissue and there is little or no induration around. The surface may be covered with sloughing material, and small areas of hæmorrhage or a small vessel which is eroded may be visible. Rarely in these early stages an acute ulceration may progress to perforation, in which case a relatively acute necrosis with but slight reactionary induration is seen. Such examples appear to occur most commonly in the course of some acute infectious complication. It is probable that nearly all anastomoses would show a certain amount of superficial ulceration shortly after operation owing to necrosis from pressure of the continuous suture, but only a few progress to the chronic stage.

Long-standing ulcers have all the characters of the chronic peptic ulcer, being deeply cut and penetrating. They tend to be circular, the mucosa is turned in and attached to the edge of the destroyed muscle, the floor is formed either of thickened peritoneum or some adherent neighbouring structure, and if sections of it are cut there is a complete absence of muscle fibre. There is much infiltration and fibrosis, at first of the surrounding wall of the stomach and jejunum, and later of adherent tissues such as the mesocolon, colon, or anterior abdominal wall. Like chronic peptic ulcers of the stomach there is usually a considerable increase of the fat of the surrounding peritoneum. They are usually single, and may be localized or extend nearly around the anastomosis. If single they are most commonly at the extremities where the afferent or efferent loop is united to the stomach (*Fig.* 17), but in some of the cases where I had performed the primary operation they were situated at the middle of the anastomosis (*Fig.* 18). The possible reason for this site has been considered in the discussion on operative technique. In some cases they are

FIG. 17.—Chronic gastrojejunal ulcer at upper margin of stoma.

FIG. 18.—Chronic gastrojejunal ulcer at centre of margin of stoma.

multiple, as many as three or four having been reported. Paterson[39] found that in 52 of his cases in which the nature of the ulcer was described, 6 were multiple.

In some cases pieces of unabsorbed suture are clearly visible passing from the floor of the ulcer (*Fig.* 19), and in others small portions of such sutures are found only on close examination. In many, however, the most careful search with the naked eye and microscope fails to reveal their presence.

Paterson[39] laid great stress upon the distinction between gastrojejunal and jejunal ulcers, and this distinction has generally been maintained. He stated that the gastrojejunal ulcers involved the gastric mucosa, whereas the jejunal were found remote from the anastomosis. He also believed that the characters of the two ulcers differed; those in the jejunum, both in appearance and clinical course, bear

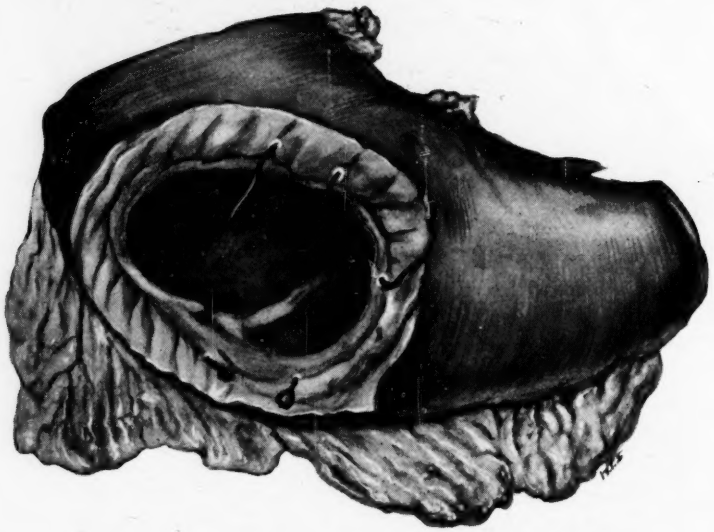

FIG. 19.—Gastrojejunal ulceration around portions of silk suture.

a close resemblance to the ordinary round ulcer of the stomach and duodenum, and they are apt to perforate before protective adhesions are formed. They are most common in the efferent limb, either opposite or just below the anastomosis. He states that the gastrojejunal ulcers lack the definite localized appearance of the jejunal ulcers and occur as an irregular ulceration around the margin of the anastomotic opening. After healing occurs the process of cicatrization may lead to partial or even complete stenosis of the anastomotic opening. It is probable that such stenosis is always due to an old marginal ulcer. These ulcers are less likely than jejunal ulcers to perforate into the general peritoneal cavity. Other observers have accepted these views, and cases have been recorded where the ulcer in the jejunum was situated several centimetres away from the margin. In my own series of 79 cases it was not found possible to make this distinction. Some of the ulcers which involved a large portion of the margin spread widely into the stomach, or extended well on to the jejunum. Even in those cases in which the ulcer appeared to be separated from the line of junction there was always an area of scarred tissue spreading from this line to the actual ulcer (*Figs.* 20, 22). In fact in no single case in the series was it possible to say definitely that the ulcer was jejunal, and therefore

the evidence at my disposal at present would lead to the belief that all such cases start at the anastomotic union.

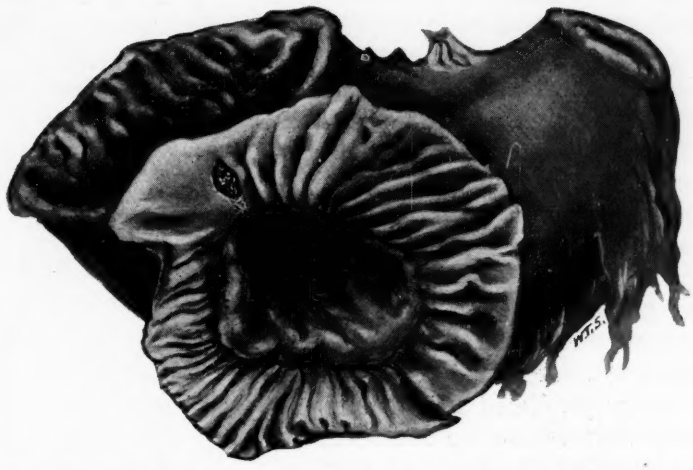

FIG. 20.—Ulcer, apparently jejunal, connected by scar to margin of stoma.

As in the case of chronic ulcers of the stomach and duodenum the ulcer in its progress tends to cause inflammatory changes in surrounding tissues, which become thickened and infiltrated. In the case of a posterior gastro-enterostomy the involved

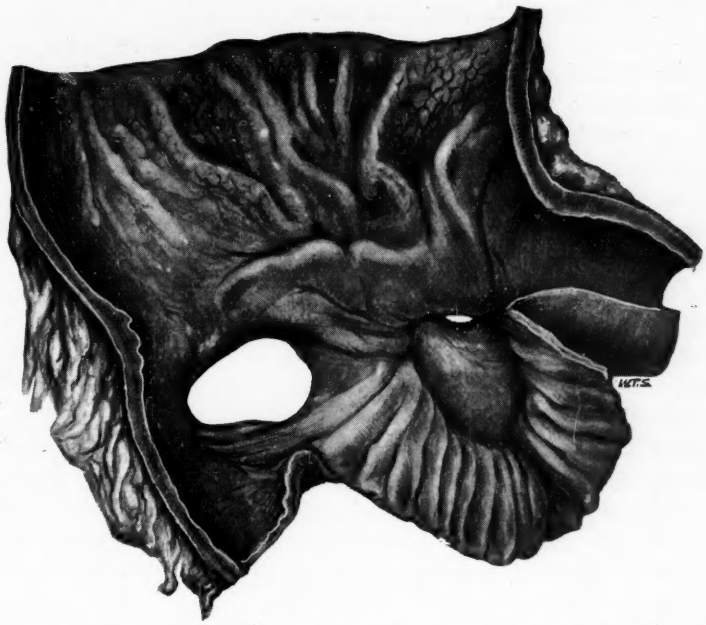

FIG. 21.—Chronic ulcer with perforation at margin of anterior gastro-enterostomy stoma.

tissue is the mesocolon, and as this becomes infiltrated it is shortened until the transverse colon is bound down and becomes firstly adherent to the base of the ulcer and then perforated, so that a gastrojejuno-colic fistula forms and the food may

pass directly from the stomach to the colon. Such fistulæ are generally only found in males, as were all four cases in my series.

In the case of an anterior gastro-enterostomy (*Fig.* 21) or of a Roux operation (*Fig.* 22) there is not the same close proximity of the mesocolon, and the base of the spreading ulcer may become adherent to the anterior abdominal wall. It may then give rise to a tender mass, or an abscess may form in the substance of the abdominal muscles. In some cases, indeed, as in one of Paterson's, the abscess may break down and an external fistula form. If the ulcerating process is rapid and acute, or if no adhesions have been formed to the surrounding viscera or to the abdominal wall, perforation into the peritoneal cavity may occur in precisely the same way as a gastric or duodenal ulcer may perforate. It is of interest to note that in the earlier published series nearly all the cases had perforated into the peritoneum or into some surrounding tissue. Thus Paterson in 1909 classifies his 52 certain cases into: (1) Perforations into the peritoneal cavity, 19 cases; (2) Non-perforations, 33 cases. These latter he again divides into two classes: (*a*) penetrating abdominal wall, 28 cases; (*b*) penetrating some hollow viscus, e.g., the colon, 5 cases.

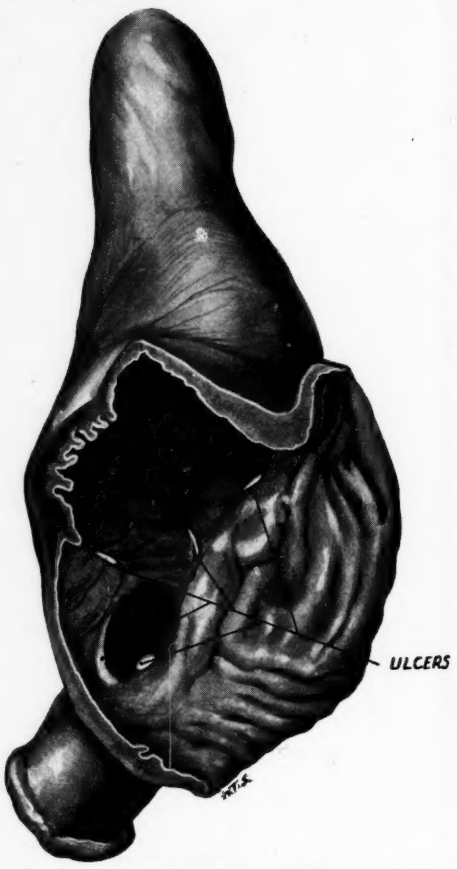


FIG. 22.—Multiple ulcers round stoma of Roux or 'in Y' gastro-enterostomy.

To-day the condition is so well recognized and the symptoms are so well known that the ulceration is as a rule diagnosed long before either perforation or penetration into a surrounding viscus has occurred. Thus in Garnett Wright's series[54] published in 1920 there were only 31 perforations in a total of 155 cases. It is interesting to note, however, that Bolton[8] in the same year recorded 4 cases of jejunocolic fistula and stated that his cases brought the recorded total of such up to 31. In my own series of 79 cases the majority were diagnosed on the symptoms of the uncomplicated ulcer, and there were in all only 6 perforations and 4 colonic fistulæ. Perforation to-day is indeed only likely to be found in the acute cases, and may occur within a few months of the performance of the gastro-enterostomy.

## ETIOLOGY

It has long been recognized that these ulcers are much more common in males, and this sex distribution seems to be more evident in the more recent series, possibly because the contributory factor of errors in operative technique is being eliminated. In Paterson's series the sex was mentioned in 50 cases, 39 being men and 11 women. Hurst states that there are about 6 males to 1 female. In my 79 cases

there were only 7 females, and in the 30 in whom I had performed the first operation only 1 was a female. There is often a family history. This is a part of the family history of primary duodenal ulcer with which all surgeons are acquainted, for Huddy[23] found in 129 cases of duodenal ulcer that 56 (42 per cent) showed a family history. Hurst, however, has shown that this inherited tendency goes deeper than the actual ulceration and that it is the variety of gastric function which runs in the family, a view which is supported by Apperly and Norris,[1] who investigated carefully a series of families comprising 86 individuals. In confirmation of this view, Hurst has been able to show that there is actually a family history of the gastrojejunal ulceration; he was able to obtain positive evidence of this in 6 out of 43 cases seen at the New Lodge Clinic, an observation which becomes of extreme interest when the cause of the marginal ulceration is considered.

As would be expected, since most of these cases follow operations for duodenal ulcer, they are generally men of middle age. Paterson's series, however, contains an interesting example in a female aged 2 months, the gastro-enterostomy having been performed for congenital pyloric stenosis.

The important questions that arise, therefore, are: Why should gastrojejunal ulceration occur practically only after the operation has been performed for duodenal ulcer? And why is it so much more common in males? Primary peptic ulcers are almost unknown in the jejunum. Occasionally one is reported which appears to be peptic in nature and is not dependent upon any specific infection. I have described an example[50] occurring in my own practice, and Richardson,[42] in reviewing the literature of this subject, reported 2 cases of his own where ulcers, apparently peptic in type, occurred 2 ft. from the flexure and about the middle of the jejunum respectively. He was able to find records of 10 other published cases. Of the total of 12, 10 occurred in men. Nevertheless these examples must be regarded almost as pathological curiosities.

Stress has been from time to time laid upon the importance of infection as a factor leading to the development of marginal ulceration. Hurst and Paterson have both discussed this point and my own experience would agree with theirs, that infection anywhere in the body is likely to increase the incidence, especially of the acute ulcers following shortly after operation. It is, however, not the sole cause, for infection is not more common in men than in women, and is not likely to be more frequent after anastomosis for duodenal ulcer than for any other lesion.

The important factor has now definitely been proved both by experiment and clinical observation to be the presence of hyperchlorhydria. Montgomery[36] found that gastrojejunal ulceration developed after gastro-enterostomy in dogs just as it may do in men. In a series of 60 such operations this form of ulceration was found in 4. Mann and Williamson,[32] acting on the assumption that the acidity was the cause of peptic ulceration, divided the pylorus, closed the proximal end of the duodenum, divided the ileum, joined the distal end of this to the pylorus, and implanted the proximal end into the ileum lower down. By this means the alkaline bile and pancreatic juices were transferred to the intestine below the anastomosis. A large number of the animals developed typical peptic ulcers. The whole of the experimental work has recently been carefully reviewed and further experimental proof provided by Matthews and Dragstedt.[34] They formed gastric pouches which were joined to the distal end of the divided intestine, the proximal end of the intestine being implanted into the ileum well below the junction of the gastric

pouch. In 6 cases where the gastric pouch was united to the ileum all developed peptic ulcers. In 13 where it was united with the jejunum 11 developed ulcers. Similar ulcers developed in gastric transplants made into the intestine low down. Such transplants secrete their own acid, the secretion, as shown by Ivy and Farrell,[25] being stimulated by gastrin bodies carried by the blood-stream. The acid acts on the mucosa long before the food could come down the intestines to neutralize it. Transplants of intestine into the stomach wall, on the other hand, never develop ulcers. The removal of the saliva by the formation of an œsophageal fistula or excision of the salivary glands was not followed by ulceration, which agrees with the clinical observation that peptic ulcers do not occur in cases of gastrostomy performed for complete œsophageal obstruction. They prove by a series of ingenious experiments that the important factor is the removal of the alkaline duodenal fluids, and of these the pancreatic secretion is the more important. When the bile was led into the stomach but the pancreatic juices were removed all the animals but one developed peptic ulcers—an interesting commentary on the operation of cholecystogastrostomy, which has been advocated by some as a cure for gastric and duodenal ulcers. Bollman,[6] however, found that after experimental obstruction of the common duct there were 64 examples of peptic ulceration and only 23 without ulcers. The earliest ulcer was found 5 days after operation and the latest 295 days. They appeared most commonly 70 to 90 days afterwards.

Somewhat similar conditions may be found in man. It is well recognized that peptic ulcer is sometimes found in a Meckel's diverticulum and is then dependent upon the presence of heterotopic areas of gastric mucosa, this condition corresponding exactly with the experimental transplants of gastric mucosa. In some cases an operation carried out for the cure of a definite lesion may, just as in the experiments, remove the juices which neutralize the acid and then be followed by peptic ulceration. I have personally reported[51] two such cases. In the one another surgeon in performing a partial gastrectomy for what was believed to be carcinoma of the stomach excised the body of the pancreas. The lesion was proved to be a simple ulcer, but the removal of so much of the pancreatic juice was followed by the formation of a large gastrojejunal ulcer. A more extensive partial gastrectomy, by further lowering the acidity, was followed by a cure. In the second case a cholecysto-enterostomy was performed for obstructive jaundice due to chronic pancreatitis. This was followed some years later by the development of a duodenal ulcer. If a lateral anastomosis is carried out in addition to the gastro-enterostomy or an anastomosis 'in Y' is performed, a condition is produced very similar to that in the experiments of Mann and Williamson, in that the alkaline secretions are made to enter the intestine below the anastomosis, and hence peptic ulceration would be expected to follow.

On the clinical side the importance of hyperacidity has also long been recognized. Paterson in 1909 investigated the acidity of the gastric juices obtained from the fistula in one of his cases and found definite hyperacidity. He discussed fully the evidence in favour of such acidity being the cause of the ulceration. It is to Hurst, however, that we owe the chief work on this subject. He shows that the frequency of marginal ulceration varies directly with the hydrochloric acid content of the gastric juice, and produces strong evidence in support of this contention. It is for this reason that marginal ulceration is almost unknown in carcinoma and is so rare in cases of ulceration of the body of the stomach, whereas with duodenal

ulcer, where the acidity is high, ulceration is more common. He found that when the gastro-enterostomy is undone for jejunal ulceration there is an exceptionally high curve of acidity, indicating that an extreme degree of the hypersthenic gastric diathesis may be a predisposing cause. The presence of such hyperacidity would also account for the familial occurrence. My own experience is in accord with this evidence. I was able to obtain the results of the test-meals at the time of the first operation in 28 of the 30 cases where I had performed this operation myself, and in 4 of the 49 where the operation had been performed elsewhere. The average figures in these 32 cases were free HCl 0·203 and total acid 73. In my own cases the average of 21 cases at the time when the marginal ulcer was found was 0·14 and 54, and in 28 of the remaining cases 0·13 and 51—figures showing a very marked hyperchlorhydria.

Of even greater interest was an investigation which I have previously published,[48] the figures of which (*Table VI*) show that the average acidity was always higher with duodenal ulcers than with lesser-curve ulcers. It was always higher in males than in females, and, what was of exceptional interest, it was highest in those cases of duodenal ulcer which later developed marginal ulceration. At that time I came to the conclusion that the development of gastrojejunal ulceration was probably due to an anatomical variation occurring in 3 to 4 per cent of men, wherein there was a wider distribution than normal of the acid-secreting cells of the stomach—a theory not very remote from Hurst's belief that there is in these people an extreme degree of the hypersthenic gastric diathesis.

*Table VI.*—AVERAGE TEST-MEALS

| | No. of Cases | Average Free HCl | Average Total Acid |
|---|---|---|---|
| Lesser curve (females) .. | 57 | 0·108 | 51·0 |
| Lesser curve (males) .. | 135 | 0·123 | 61·0 |
| Duodenal (females) .. | 44 | 0·136 | 57·6 |
| Duodenal (males) .. | 213 | 0·165 | 63·5 |
| Duodenal, later gastro-jejunal .. .. | 13 | 0·190 | 71·0 |

On these considerations it seems permissible to accept the following: (1) Gastrojejunal ulceration is only common after gastro-enterostomy for duodenal ulcer; (2) It is much more frequent in males; (3) It occurs after 3 to 4 per cent of all gastro-enterostomies for duodenal ulcer; (4) It is directly due to hyperacidity; (5) The hyperacidity is a congenital and possibly a familial fault.

## SYMPTOMS

It has already been mentioned that in the earlier published series the condition was usually not diagnosed until perforation or penetration had occurred. In the more recent series these complications are much rarer, for the symptoms of non-penetrating ulcers are now well recognized and the condition treated. It is probably because of these earlier series that the statement is still so often made that there is frequently a long latent period, perhaps of many years, before symptoms arise. Such a history is indeed often obtained to-day in a patient who is first seen

when the ulcer is well established. Although such patients will state that for years after the operation they are quite well, the one fact that has been impressed upon me by watching them carefully in a Follow-up Department is that there is generally no latent period. A patient with a gastro-enterostomy for a duodenal ulcer is, as a rule, one of the most satisfactory of our cases. He is soon able to eat all food with no thought or care, and is able to carry out his normal work. His usual remark is that he has never had a day's discomfort since the date of his operation, and his presence in the Follow-up Department does much to encourage the surgeon in his many disappointments. The patient who later develops a marginal ulcer does not as a rule make this satisfactory progress. He is well, but has a little flatulence or he has to be careful of his food; he occasionally has nausea. He cannot undertake heavy work or he has to continue with his bismuth and alkali long after the usual period, or symptoms will return. I have learnt now to regard all such cases with considerable mistrust, especially if they had an unduly high acidity before the operation. They will generally develop characteristic symptoms of marginal ulceration, which may not appear for some years, at which time they will have forgotten their earlier minor symptoms. In the 30 cases of my own series the average time elapsing between the gastro-enterostomy and the onset of definite symptoms was 2 years and 11 months, but in two cases it was only a month, and in one was as long as 14 years.

Clinically there are two definite groups. In the first the dominant symptom is hæmorrhage. This may commence as a mild hæmatemesis or melæna shortly after operation, or the patient may continue in a very fair state of health for a year or more and then have a sudden severe loss of blood, generally in the form of melæna. In the earlier cases this symptom was often regarded as evidence of persistence of the duodenal ulcer, but, as I have frequently stated, I have never yet seen a duodenal ulcer persist after an adequately performed posterior gastro-enterostomy. I believe that all such cases have marginal ulceration, but as at this stage it may be only acute, it may be readily overlooked at operation or even indeed at a post-mortem examination. In my series of 79, 32 had had hæmorrhage, but in many of these it occurred later when other symptoms were present.

In the more common variety there is in the earlier stages only a minor degree of discomfort, fullness, or flatulence, which is later replaced by a definite pain, at first thought to be that of the old duodenal ulcer. It simulates the original lesion in that it becomes periodic, so that the patient may have severe attacks lasting for two or three weeks and may then be quite free for several months. It is severe, occurs late after food, is relieved by food, and often wakes him at night. A careful investigation will show, however, that the pain differs from that of a duodenal ulcer in that it is often referred to the lower abdomen, and especially to the left iliac fossa, and that vomiting, which is so rare a symptom with uncomplicated duodenal ulcer, is often present and generally gives transient relief to the pain. In the latter stages, as the ulcer penetrates, the pain becomes more and more severe and much more constant. It may indeed become agonizing and the patient pass into a stage of deplorable misery. In my 79 cases pain was present in 73, and it must therefore be regarded as the most frequent and characteristic symptom. Vomiting, generally giving temporary relief, was present in 46 cases. In the earlier stages the patient retains his appetite and the test-meal shows a high acidity. Later when the pain becomes more severe his appetite may begin to fail.

The value of X-ray investigation has been much disputed. There is no doubt that in the earlier stages very little can be seen, and even in the later stages considerable skill is required to interpret the findings. The presence of tenderness over the visualized stoma is a finding of considerable value, as are hyperactivity and rapid emptying. Irregularities in the margin of the stoma and above all the presence of a well-marked meniscus close to the stoma confirm the diagnosis (*Fig.* 23). In my own series the very skilful work of Dr. Vilvandré has been of great help, and he was able to make a positive diagnosis in a high percentage of the cases.

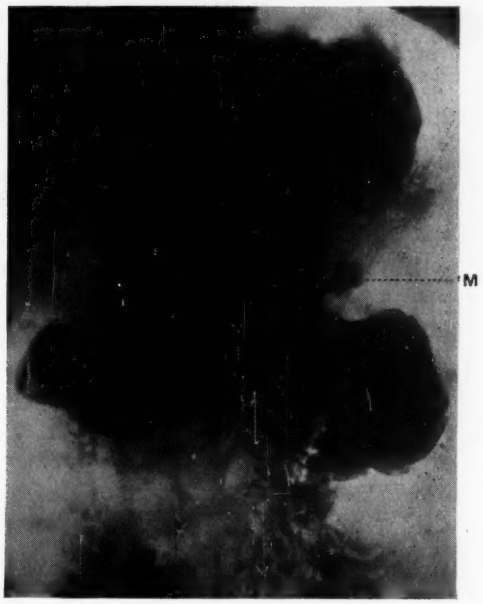


FIG. 23.—X-ray showing meniscus (M) of large gastrojejunal ulcer.

Of the symptoms of perforation nothing need be said, for they differ in no way from the symptoms of a similar complication of gastric or duodenal ulcer.

When penetration has occurred and a fistula passes into the colon the clinical picture may be considerably altered, the cases generally falling into one of two groups. In the one they are dependent upon the presence of a free opening between the stomach and colon. According to Bolton, if the ulcer had been gastro-jejunal, the opening between the two viscera is direct and the dominant symptom is fæcal vomiting and the eructation of foul gas. If, however, the ulcer had been jejunal, the communication will be indirect through the jejunum, in which case the chief symptom is diarrhœa, due to irritation of the colon from the presence of the gastric contents. In practice it is, however, difficult to make this distinction. It is true that in some cases the presence of colonic content in the stomach with foul vomiting, eructation, loss of appetite, and wasting are the chief symptoms, but even then attacks of diarrhœa are frequent and the patient feels his best when constipated. If the colonic symptoms are more marked, there will generally be found some evidence of gastric regurgitation and gastritis. In either case the examination of the test-meal will reveal the presence of fæcal material, but the certain diagnosis to-day rests upon the X-ray examination. The barium meal will be found to enter the colon almost at once, or a barium enema will pass through into the stomach. In such patients the pain is very varied and often the severity seen with the original ulcer seems to be diminished, probably because of the neutralization of the acid by the colonic contents or its diminution owing to the resulting gastritis. Hurst has made the observation that with the formation of a fistula the ulcer usually heals. In some cases, however, the pain remains severe. The patients are usually wasted and often indeed emaciated.

In the second group the dominant symptom is one of intestinal obstruction, for it will nearly always be found that the fistula is associated with much fibrosis, which will spread around the colon and thus narrow its lumen. In these patients

there will be attacks of colicky pain with dilatation and visible peristalsis of the proximal colon, but even in this group attacks of fæcal vomiting may occur, and the patients are nearly always very wasted. The X-ray investigation, as in the last group, will generally reveal the presence of a fistula as well as the obstruction.

When the anastomosis has been anterior the penetrating ulcer usually becomes adherent to the anterior abdominal wall, generally behind the left upper rectus. Rigidity and tenderness of this muscle may be seen in the earlier stages, and later a swelling develops in the substance of the muscle. If left untreated, this becomes red and œdematous, and later breaks down so that a gastric fistula is formed through which much of the gastric contents may escape and lead to digestion of the skin. Like the colonic fistula, these are becoming rare to-day, as treatment is usually undertaken before their inception.

## TREATMENT

As with the original peptic ulcer, there is considerable divergence of opinion between physicians and surgeons concerning the value of medical treatment. Hurst[24] states that with careful medical measures gastrojejunal ulcers can be cured, but he also very fairly remarks that if the regulations are disobeyed the ulcer tends to recur, and that if severe obstruction is present or prolonged medical treatment fails, resort should be had to surgery. Of 25 of his cases treated medically, 13 were more or less completely relieved. My own experience of medical treatment has, however, been very unsatisfactory. The aid of the physician has always been sought in the earlier stages of this condition occurring in my own practice, but wherever the diagnosis could be considered as certain the lesion has always progressed and surgery has later been necessary. In every other case where the first operation had been performed elsewhere medical treatment has been tried before my interference had been sought, but it is only fair to state that of necessity it is only the failures which are sent to me, and I have therefore no means of knowing how many may have been cured.

If surgical treatment is required, Hurst believes that the best as well as the simplest operation is local excision. Such is not my experience. In my earlier cases, acting on the belief that these ulcers were generally dependent upon the use of unabsorbent sutures, a local excision was performed and the opening re-sutured with catgut. There were 13 such cases, of which 1 died after operation. Of the remaining 12, 6 recurred and further operation was required. It is true that some of these patients, and especially those with a colic fistula, are extremely ill and will be able to stand only the simplest operation. In these cases separation of the fistula with local excision of any ulcer and closure of the openings may alone be possible, but such steps should always be regarded merely as temporary measures to save the life of the patient. He must be carefully treated and watched afterwards, and, if the ulcer persists, the more complete operation performed when his condition has improved.

The operation which at one time was very popular and still has its advocates is excision of the ulcer, separation of the anastomosis, and closure of the two openings, thus restoring the condition to normal. In some cases there is so much stenosis of the duodenum from healing of the old ulcer that such a step is only possible if a gastroduodenostomy or some similar step is performed at the time.

This operation is, however, theoretically unsound, as it restores the tendency for the formation of a duodenal ulcer and there is no reason to believe that after the passing of time the hyperchlorhydria is diminished. Hurst lays stress upon this point and states that if the opening is closed a fresh ulcer may develop. There were 2 examples in his series of 44 cases. One case of such recurrence after closure of the opening by another surgeon occurred in my series, but I have no statistics of its frequency, for I have never performed the operation.

The method which would seem to offer the most satisfactory results is that of partial gastrectomy, for it has been shown that this operation is followed by a much lower incidence of marginal ulceration than is gastro-enterostomy. It is, however, an operation of very considerable severity. Much time may be occupied in freeing adhesions and making the anatomical relationships manifest before the actual resection is commenced. The operation becomes in fact one of the most exacting and difficult procedures within the abdomen. Being performed, as it is so frequently, on patients who are often exhausted by long-continued severe pain, by vomiting, by hæmorrhage, or by malnutrition from the presence of a fistula, it is likely to be associated with a high mortality. For this reason Hurst condemns it and states that it is only advisable in very exceptional conditions. He quotes von Eiselsberg[45] as having a mortality of 25 per cent for such operations. He also quotes Klein[27] as finding that only 2 out of 6 of these operations were followed by achlorhydria, and therefore suggests that the risk of a new ulcer remains in 66 per cent. There is no doubt that in the earlier operations performed by all surgeons the mortality is high, but as experience is gained in the selection of cases for operation and the performance of temporary palliative operations upon those who are dangerously ill, this can be greatly reduced. Finsterer[15] had a mortality of 9 per cent in 76 cases. In my own series of 79 cases a partial gastrectomy was performed in 61, and 16 of these patients died, a mortality of nearly 25 per cent, but most of these deaths occurred in the earlier cases before experience was gained, and in the last series of 20 cases there was only 1 death, a mortality of 5 per cent. The risk of recurrence after this operation is very slight. Finsterer states that if three-quarters of the stomach is removed recurrence never takes place. In my own series of 61 cases there has only been 1 recurrence, this being the one already described, where owing to adhesions too small a portion of the stomach was removed. A later and larger resection was followed by a complete cure.

It would therefore seem reasonable to accept the view that partial gastrectomy, although not the operation of choice for duodenal ulceration, is so for gastrojejunal ulceration, and should only be replaced by more simple methods if the condition of the patient will not warrant its use.

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# PROTRUSIO ACETABULI (CENTRAL LUXATION)

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During the last few years considerable interest has been manifested in American literature in the condition of intrapelvic protrusion of the acetabulum, as shown by a number of articles contributed to the journals.[1-6] Prior to this references to the condition are almost entirely restricted to Continental literature, especially the German.

Approximately fifty-one true cases have been described in one hundred and ten years since Otto's original article in 1824.[7] The following ten cases are added as likely to be of interest, although there is reason to believe that the condition is not so rare as the above figures would indicate. Fifteen cases in all were found in a survey of 298 radiographs of pathological hip-joints, all of which could have been labelled with a broad diagnosis of rheumatic hip—the patients having been sent by their doctors to a rheumatic clinic. That is an incidence of 5 per cent in this type of patient.

## ETIOLOGY AND CLASSIFICATION

In the literature one finds little agreement on the subject of etiology, yet it is mainly on the supposed etiology that attempts at classification have hitherto been made. No fewer than sixteen primary causes have been held responsible by the various authors in their reports on cases, and possibly this total is incomplete. This has given rise to a wide variation in the ultimate diagnoses.

In view of the confusion at present existing, and admitting that protrusion is a disease process, not an entity, I should like to propose a simpler classification, based on the etiology where it is known, but more particularly on the radiographic findings: (1) A group conforming to the hypothesis of Eppinger,[14] or some theory of growth disturbance; (2) The rheumatic group, (*a*) specific infection (gonococcal), (*b*) non-specific infection, (*c*) metabolic arthritides; (3) A group including many varieties due to gross destructive disease.

**Group 1.**—The infantile acetabulum is the site of union of the three pelvic bones, by the medium of a tri-radiate cartilage, in which ossific centres appear in the twelfth year and union commonly takes place about the sixteenth year.

Eppinger considered that the cases he described were due to a disturbance of growth affecting this Y-shaped cartilage, possibly a process resembling the more recently described osteochondritis.

The etiology of this process of malacia has not been explained satisfactorily. There is no direct evidence to support this theory, but equally it is not likely to be disproved.

On the evidence of the history and radiographic findings, I suggest that, for a small group, Eppinger's theory, which has never been accepted, is correct.

The radiograph of this type shows a joint with an intact cartilage and no evidence of infective changes in articular bone. The acetabulum protrudes to a varying extent, and on this depends the degree of limitation of movement, which may be so slight that the patient is not aware of defect,[15] or, alternatively, the loss of full range of movement, especially abduction, which she notices, is insufficient to make her seek treatment (*see Figs.* 24, 25). At a later stage, owing no doubt to the mechanical disadvantage at which the joint is working, osteo-arthritic and infective changes are often superimposed, and the patient now complains of definite pain; or, by reason of these changes, which produce osteophytes and loss of cartilage, or both, the patient is aware, for the first time, that movement is restricted.

The histories which *Cases* 1 and 2 gave also point to a non-inflammatory origin in early life:—

*Case* 1.—The patient gives a clear history of limitation of movement since the age of 15; she remembers that when she was learning to swim the instructor remarked on the fact that she was unable to separate her legs so as to perform the 'frog-leg' kick of the breast stroke—her legs "trailed behind her"; she had no pain at this time. For the last twenty years, however, she has had pain in the knees and the lower back, which may have been referred from the hips. She first noticed definite pain in the left hip four months ago after a fall. The condition is bilateral, but she has no pain on the right side. (*Fig.* 24.)

FIG. 24.—*Case* 1. Left hip. Female, age 62 years. Bilateral. Duration forty-seven years. Limited movement forty-seven years. Pain in left hip four months. Right hip no symptoms except limited movement. No pain on this side, but condition identical. Cartilage intact. No arthritic changes. No other joints involved. (*Case of Dr. Copeman.*)

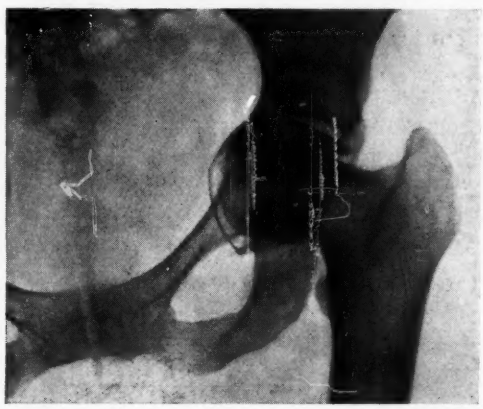

FIG. 25.—*Case* 2. Left hip. Female, age 40 years. Bilateral. Duration twenty-four years. Limited movement twenty-four years. Pain in right hip, and to a lesser extent in left hip, for nine years. Cartilage thin at the point of pressure, otherwise intact. Floor of acetabulum extremely thin. No evidence of infective changes in bone or joint. Osteophyte formation commencing. No other joints involved. (*Case of Mr. Verrall.*)

*Case* 2.—The patient noticed she was unable to obtain the full movements of the hip-joints when 16; she then found she was unable to cross her legs when seated, and she has been aware of limitation of movement since that time. (*Fig.* 25.)

*Case* 3.—This case is placed in this group on radiographic evidence alone. The patient first noticed pain and stiffness in the hips four months ago; she was not aware of limited movement before this. This might be explained by the fact that, although the head is protruding, the neck has not been buried. Radiographically, the most obvious obstruction to movement is the presence of osteophytes on the femoral head, and it is difficult to imagine that these have developed in four months. Otherwise, this case resembles *Case* 2. (*Fig.* 26.)

Amongst others, the cases of Frölich[15] could be classified in this group. Similarly Hertzler[16] has reported a case aged 29 years with a history of ten years' duration. Recently Reed[6] has published the case of a nurse aged 24 years with the onset of disability at 16 years.

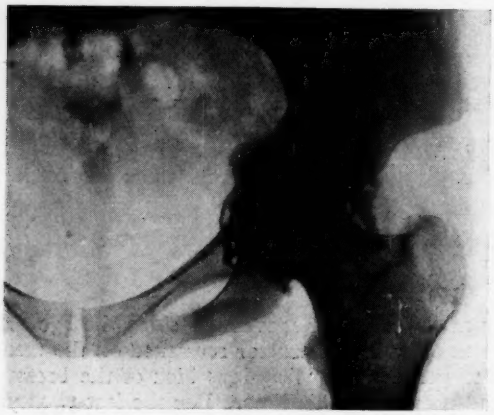

FIG. 26.—*Case* 3. Left hip. Female, age 33 years. Bilateral. Duration stated as four months. Onset of pain and limitation of movement simultaneously four months ago. Acetabulum the thickness of paper. A very large osteophyte has formed on the head of the femur. No other joints involved. Similar condition to *Case* 2. (*Case of Dr. Heald.*)

The conclusion is, therefore, that there is a small group of cases which is compatible with a mode of origin such as Eppinger has described, or with some similar theory.

The protrusion, having formed, does not appear to increase in size. *Case* 2 is in substantially the same condition as in 1929,[3] except that osteophytes are now forming and the cartilage on the right side is becoming thin; the protrusion, however, is no greater. In view of the presence of an intact cartilage in the early stages and the absence of sclerosis of bone or other signs of infective changes, the term 'arthrokatadysis' of Verrall is very suitable for the group. Later the combination of trauma and superimposed infection, to which the joint in this state appears susceptible, destroys the characteristic features, and it is not possible to differentiate this group from the following.

SUMMARY.—Summarized, these points are:—

1. A history of limited movement, especially abduction and adduction, the onset being in adolescence and painless.

2. Since the limitation of movement depends on contact being made on the rim of the acetabulum by the femur, the minor varieties may be overlooked, until rim osteophytes further limit movement or infection causes pain.

3. The protrusion appears to be non-progressive, the cartilage is of normal width, and the acetabulum floor very thin. The articular bone is unaltered in appearance. Later, infective arthritic changes tend to be added; these changes are progressive.

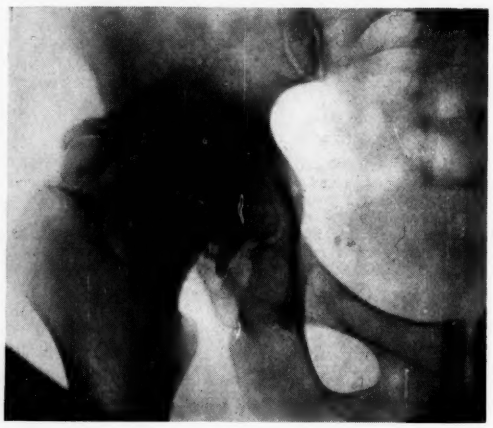

FIG. 27.—*Case* 4. Left hip. Male, age 42 years. Unilateral. Duration eighteen years. Circumferential thinning of cartilage. Sclerosis of femur head and acetabulum. Secondary osteophytes. No other joints involved. In spite of the radiographic appearance, the patient denies any hip trouble before the acute onset—was a regular member of a football team and had played all games.

**Group 2 (Rheumatic Group).**—Certain evidence has been produced in the past which favoured the gonococcal origin of some of these cases, notably that found in the writings of

Schlagenhaufer,[8] Henschen,[9] Kienböck,[10] Breus,[11] Chiari,[12] and Zwicker.[13] Of the five male patients in this series, four had had gonorrhœa. From this point of view the histories are summarized :—

*Case* 4.—Age 42 years. First noticed pain in the left hip at 24 years of age ; acute onset of severe pain ; was in hospital for five months ; had contracted gonorrhœa eight weeks before onset of pain ; no previous disability of hip. No other joints involved. Unilateral. (*Fig.* 27.)

*Case* 5.—Age 63 years. Pain began in 1916 in the left hip. It was of moderate severity, and the first attack lasted a week ; it then became intermittent. He was admitted to hospital in 1919 for seven weeks for hip trouble. Noticed pain in the other (right) hip four years ago. Had gonorrhœa twice between 1904 and 1909. Bilateral changes. No other joints involved. (*Fig.* 28.)

FIG. 28.—*Case* 5. Left hip. Male, age 63 years. Bilateral. Duration seventeen years left hip, and four years (?) right hip. Dense sclerosis of femur head with cavitation of bone ; the outline is irregular and pointed. Osteophytes present. More recent radiographs show loss of cartilage. No other joints involved. (*Case of Dr. Nisse.*)

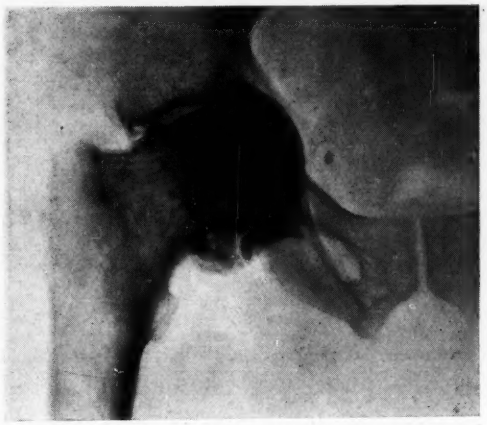

FIG. 29.—*Case* 6. Right hip. Male, age 64 years. Bilateral. Duration forty-four years. Sclerosis of femur and cavitation. Thickened and sclerosed acetabulum. Cartilage absent except outer and upper aspect of joint. (*Case of Dr. Ray.*)

*Case* 6.—Aged 64 years. Pain in hips at 20 years of age ; limped slightly when walking ; no acute attacks, constant pain now. Gonorrhoea at 20 years. Bilateral. No other joints involved. (*Fig.* 29.)

*Case* 7.—Aged 67 years. Pain began about seven to eight years ago, as far as he can remember ; was first noticed after a minor fall. Had gonorrhœa in 1925, and is being treated at present for stricture. Is not sure of the time relation between infection and hip pain ; is mentally dull and unreliable. Bilateral. No other joints involved. (*Fig.* 30.)

The coincidence of arthritis with venereal infection in these patients suggests that the latter may have had some casual relationship to the joint lesions.

*Case* 8 (the fifth male in the series).—The patient was a man aged 44 years, with very advanced spondylitis of the so-called Marie-Strümpell type, with complete calcification of the intervertebral ligaments and obliteration of the sacro-iliac synchondrosis. There was no history of gonorrhœa, and the condition was bilateral. (*Fig.* 31.)

In the histories of the females of the series the gonococcus could not be excluded at this late stage with any certainty ; few definite factors were discovered which appeared to bear direct relationship to the hip disease. One case suffered

from polyarthritis, the onset being at the menopause. Radiographs of many joints were obtained and the appearances were those of a metabolic rather than an infective arthritis. Two cases (*Figs.* 32, 33), aged 67 and 76, had a subacute onset with multiple joint involvement and were considered non-specific infections.

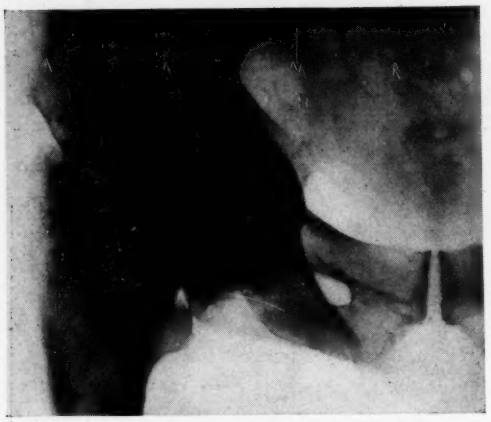

FIG. 30.—*Case* 7. Right hip. Male, age 67 years. Bilateral. Duration about eight years. Femur head dense with irregular outline and has protruded to the limit of the neck. The cartilage is very thin. The floor of the acetabulum is sclerosed. Osteophytes are present. (*Case of Dr. Copeman.*)

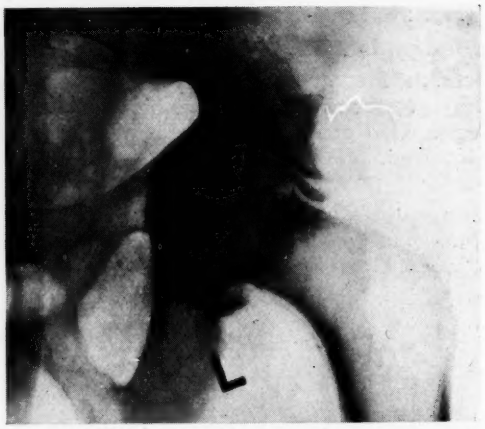

FIG. 31.—*Case* 8. Left hip. Male, age 44 years. Bilateral. Duration ten years. Onset of spondylitis twenty years ago. Cartilage thinned. Little or no reaction in bone of head of femur. Osteophytes present. Calcification of intervertebral ligaments and obliteration of sacro-iliac synchondrosis. (*Case of Dr. Ray.*)

FIG. 32.—*Case* 9. Left hip. Female, age 67 years. Unilateral. Duration stated as one year. Head of femur sclerosed and osteophytes present. Cartilage absent except outer aspect of joint. Acetabulum sclerosed. In left knee and right wrist pain and swelling. Gross dental sepsis. (*Case of Dr. Howitt.*)

FIG. 33.—*Case* 10. Right hip. Female, age 76 years. Unilateral. Duration one year. Irregularity of articular surface of femur. Sclerosis and thickening of acetabulum. Generalized loss of cartilage. Osteophytes developing. Pain and swelling of left wrist two to three years. Gross dental sepsis. (*Case of Dr. Nisse.*)

From the evidence of these cases, it can be inferred that the etiology of this group is the etiology of chronic rheumatic affections in general, and a specific cause to explain all cases is lacking.

It is not possible to describe an exact picture that will include all the characteristics of this rheumatic group; it is typical, however, that the cartilage is absent or thin, and this thinning is generalized over the circumference of the femur head, not at the point of maximum pressure only, as in early senile osteo-arthritis. The head of the bone is often heavily sclerosed, with minor irregularities in outline, and occasionally somewhat pointed. New bone is laid down as a compensatory process over the bulging acetabulum, a feature which is absent from *Group* 1, except in the later stages. The wall is therefore more thickened and dense in *Group* 2 than in *Group* 1.

Possibly the degree of protrusion in *Group* 2 depends to some extent on the virulence of the infective organism. Similarly, it would seem to be necessary for these infective changes in the acetabulum to be in advance of any destruction which may take place in the femur head. *Case* 4 shows that a virulent infection alone will not necessarily produce marked protrusion; in this patient a softened and flattened femur head has made the result less marked than otherwise might have been expected. Osteophytes develop in this form of the disease, especially at the base of the femur head. The head may itself show cavitation, as is found so frequently in the common osteo-arthritic changes (morbus coxæ senilis) which affect this joint.

There is now a tendency to classify all cases of intrapelvic protrusion as osteo-arthritis. This is correct only as a statement of end-result. Admittedly in some cases osteo-arthritis is the only possible diagnosis; occasionally, however, by a careful history and correlation with X-ray appearances, the origin of the deformity may be traced.

In some cases of this series the fact has been observed that little reliance can be placed on the patient's estimate of the duration of the arthritis when the history given is short. Many of the changes in the radiographs could not possibly have been produced in the brief period of the patient's complaint. *Case* 1 considered her trouble began four months ago, when she first suffered pain. She was neglecting the fact that she had had limited movement for nearly fifty years.

**Group 3.**—The third group is a heterogeneous collection of little interest, the condition being often something in the nature of an accidental occurrence in some gross disease of bone, as for example when secondary neoplasm[20–23] or echinococcal[24] disease of bone involves the acetabulum, or a part of the destruction produced by pyogenic organisms,[25] syphilis,[26] and tuberculosis.[27, 28]

It would simplify descriptions if this group were excluded from the reports of true Otto pelvis.

I am greatly indebted to the Physicians of the British Red Cross Clinic for Rheumatism, Drs. Copeman, Heald, Howitt, Nisse, and Ray, for the generous manner in which they have allowed me to use their cases; to Mr. Verrall for permission to radiograph his case; and to Dr. Gilbert Scott for the use of the radiographic material of the Clinic.

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# BONE-GRAFT FOR NON-UNION OF THE CARPAL SCAPHOID*

By GORDON MURRAY, Toronto

When a patient complains of a 'sprained wrist' with symptoms lasting more than two weeks, the possibility of a fracture of the carpal scaphoid should be considered and the necessary investigation undertaken. If examination shows a fair range of movement of the wrist-joint in all directions, but limitation at the extremes of all movements, with tenderness in the anatomical snuff-box when the hand is adducted, and tenderness over the dorsal and palmar surfaces of the scaphoid, with negative findings elsewhere, it is probable that there is a fracture of the scaphoid. If after an injury causing symptoms in the wrist-joint these findings persist for months or years, the possibility of non-union of a fractured scaphoid should be considered.

X-rays taken of the carpal scaphoid in the antero-posterior and oblique directions usually provide the necessary evidence, but recent fractures may be difficult to see with X-rays taken in various directions. There are cases of recent fracture in which a clinical diagnosis is easily made but X-rays do not show the lesion at that time ; yet plates taken a few weeks later show obvious signs of fracture. Older fractures with non-union are seen more easily, owing to the early rarefying osteitis and fibrous union, and later sclerosis of the adjacent margins of the fragments.

Fractures of the tuberosity always unite if the fragments are in apposition, and those through the intra-articular surfaces about the waist of the bone, which are the most common, may unite if treated. Johnson[1] has shown experimentally that there is an adequate blood-supply to the bone as a whole, and to both fragments, in case of fracture. He showed also that the type of reaction in fracture of the scaphoid is similar to that in other bones, but is more localized to the fracture line, as there is so little periosteum to assist in the formation of subperiosteal callus. The formation of bone in the callus is slower than in other fractures.

Adams and Leonard[2] say that untreated fractures of the scaphoid always result in non-union, and with the usual form of treatment a small percentage shows union. Grace[3] supports this view with a report of cases showing a high percentage of non-union in those treated by immobilization for a period varying from six to eight weeks. Böhler,[4] on the other hand, states that most fractures of this bone unite if treated early by prolonged fixation, and this agrees with our results. However, there are many injured wrists treated as sprains without fixation which provide the cases of fracture with non-union.

Certain anomalies should be kept in mind to prevent confusion in making a diagnosis of fracture and non-union. Ordinarily the centre of ossification appears in the sixth year, but there may be two centres and these may not unite, but may persist to form two bones. Rarely the os centrale may persist as a third bone.

* From the Department of Surgery, University of Toronto.

Methods of treatment that have been advocated for non-union are excision of one or of both fragments or of all the bones in the proximal row, including the fragments of the scaphoid. Any one of those operations leaves a deformed wrist with some permanent disability in the form of impaired function and frequently with pain on active use of the hand (Grace[3]).

If the individual with non-union of a fractured scaphoid is able to follow a sedentary occupation and avoid energetic use of the hand, he may have little discomfort; but if heavy labour, sport, or full range of movement of the wrist is important, the best prospects are offered by a bone-graft. There is a report of one case[2] in which this operation was used, but it has not come into general use. In our cases with non-union this form of treatment has yielded the best results. It is essential to have X-ray evidence that both fragments are viable and in apposition, and that there is no arthritis.

## OPERATIVE TECHNIQUE

With the hand in full adduction, a curved incision is made along the radial surface of the wrist-joint extending about $1\frac{1}{4}$ in. upward and downward from the radial facet of the scaphoid. The ends of the incision are curved towards the posterior surface of the wrist, and the convexity anteriorly should reach the tendon of the abductor pollicis longus. The radial nerve and vessels and the abductor tendons of the thumb are retracted anteriorly, and the extensor pollicis longus tendon posteriorly. This provides exposure of the tuberosity of the scaphoid. A small transverse opening is made through the dorsal capsule of the wrist-joint, exposing the dorsal surface of the radial facet of the scaphoid, and on this surface the fracture line is apparent. If the other bones of the carpus have not been disturbed by the injury, the fragments of the scaphoid will not be displaced, and in that case the fracture line is not disturbed by curetting, etc. After clearing the most prominent area of the tuberosity, a small nick is made in the bone at this point with rongeurs, in order to provide for countersinking of the graft and prevention of bone proliferation, which might interfere with abduction of the wrist-joint.

With a bit about $\frac{5}{16}$ in. a hole is drilled, beginning at the nick in the tuberosity, through the proximal fragment, across the fracture line, and into the distal fragment. Great care is necessary to line the drill properly, assisted by observations through the dorsal window, so that no cartilaginous surface is damaged. The depth of the drill hole, after the fracture line is crossed, should be measured every few millimetres to prevent damage to the semilunar facet of the scaphoid by going too far.

A suitable piece of cortical bone is removed from the tibia and shaped to fit snugly. It is passed well through into the medial fragment, taking care that the fragments are not separated, and is cut to leave no projection. The dorsal ligament is repaired.

The hand is supported in a circular plaster in a cock-up position for eight weeks. After this period all our cases in the general hospital had X-ray evidence of bony union, and within a few months the fracture line had disappeared. There was complete restoration of function, with a full range of movement in all directions without pain and with normal grip.

## CASE REPORTS

*Case* 1.—B. S., aged 31, painter. Admitted on Jan. 27, 1931, with a complaint of pain and stiffness of the right wrist for two months. This began with an injury received when cranking a car, and persisted with no improvement of symptoms. A diagnosis of sprained wrist was made by his family physician, and was treated by bandaging.

On examination there was no deformity and no swelling. There was limitation of flexion, extension, adduction, and abduction, with marked pain on active and passive movements beyond this range of movement. The grip was weak, and increased force applied in any way caused pain in the wrist region. With the hand adducted there was marked tenderness over the radial facet of the scaphoid, just distal to the styloid process of the radius. X-rays (*Fig.* 34) showed a fracture through the waist of the scaphoid, without displacement, and both fragments apparently viable.

FIG. 34.—*Case* 1. X-ray taken two months after fracture of carpal scaphoid, showing non-union.

A bone-grafting operation was undertaken, and *Fig.* 35 shows the graft in place, bridging the fracture line and lying in contact with both fragments. *Fig.* 36, taken four months after operation, shows solid union of the graft to both fragments and disappearance of the fracture line. The circular plaster extending from the elbow region to the metacarpo-phalangeal joints was replaced in ten days by a more snugly fitting one, which was left on for eight weeks. X-rays taken at this time showed signs of union, but firm union as shown in *Fig.* 36 was evident after a period of four months. The post-operative course was uneventful. Limited activities of the wrist-joint were allowed

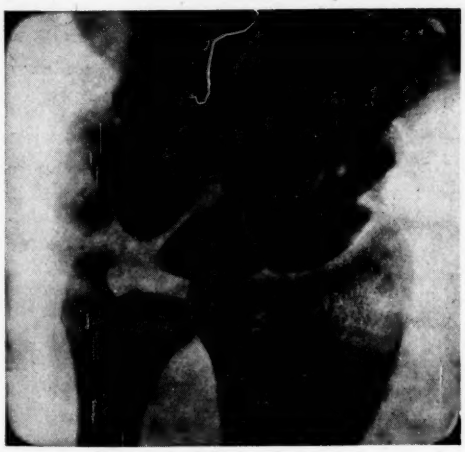

FIG. 35.—*Case* 1. X-ray taken immediately after bone-graft operation, showing graft in position.

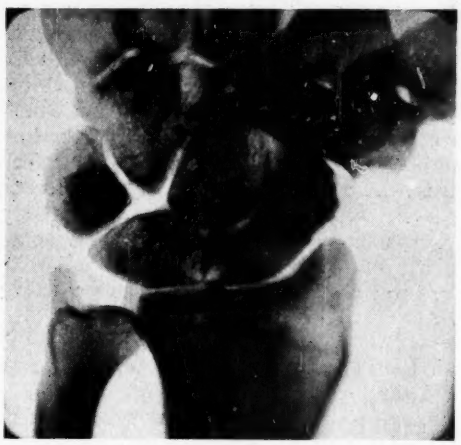

FIG. 36.—*Case* 1. X-ray taken four months after bone-graft, showing solid union of fragments.

after removal of the cast at eight weeks, and full range of movement was present without discomfort after fourteen weeks. The patient returned to his usual occupation of painting and decorating four months after operation. On clinical examination there was no disability of the wrist-joint, and this has remained unchanged over a period of two and a half years.

*Case* 2.—F. S., aged 23, medical student. Admitted on June 2, 1931, with pain and disability of the right wrist-joint of four months' duration. A fall during a game of rugby caused some pain and stiffness of the wrist-joint which persisted. An X-ray taken on the day of the injury did not give conclusive evidence of a fracture. After four months' treatment by bandages and wrist supports by the family physician, X-rays showed a fracture with well-marked rarefying osteitis, with both fragments viable, as shown in *Fig.* 37. On June 4 a bone-graft was placed across the fracture line. The small proximal fragment made the operation technically more difficult, but it was carried out without injury to articular cartilage (*Fig.* 38). The wrist-joint was fixed in plaster for eight weeks, and in four months solid union of both fragments and graft had taken place (*Fig.* 39). There was complete recovery of function of wrist-joint, with full range of movements in all directions without discomfort, and the patient was able to return to regular sports on Dec. 1, 1931.

*Case* 3.—H. C., aged 32, fireman. Feb. 20, 1932, a fall on the outstretched left hand caused severe injury to the wrist. This was treated by his family physician as a sprained wrist for about one month without improvement in symptoms. Examination at this time showed no deformity, no swelling, but very great limitation of all movements, and tenderness in the anterior, posterior, and lateral surfaces of the scaphoid. X-rays showed a typical fracture through the waist of the bone, with no displacement, with both fragments viable, and with no signs of union.

At operation on March 24 a small groove was cut across the fracture line, which showed no signs of union, and a small graft of bone from the tibia was fitted in place (*Fig.* 40). After fixation for eight weeks, the plaster was removed and X-rays were taken. There was some X-ray evidence of union, so the arm was left without support and movements were encouraged. The graft and fragments united solidly and a full range of movement without discomfort was obtained. The patient returned to his regular occupation as fireman without disability, and has continued at this work for more than one year.

*Case* 4.—F. P., aged 33, labourer. On May 18, 1932, both wrists were injured, a Colles's fracture of the left, and a fracture of the scaphoid on the right. With the usual treatment the Colles's fracture united and a good result was obtained, but the scaphoid continued to cause trouble. Light work was resumed for a short period during September and October, but was discontinued on account of pain in the right wrist. Examination on Dec. 21 showed some swelling in the region of the wrist-joint with painful limitation of all movements, and tenderness over all surfaces of the scaphoid. X-rays showed a fracture through the waist of the bone, without displacement, with no signs of union, and with both fragments viable.

At operation by Dr. W. E. Gallie on Dec. 22 an incision across the anatomical snuff-box exposed the scaphoid with the fracture line. There was firm fibrous union, but no evidence of bony union of the fragments. A drill-hole was made to cross the fracture line and a graft from the tibia fitted in place. The fracture surfaces were not disturbed.

Primary healing occurred, and the plaster support was removed on Feb. 23, 1933. X-rays showed evidence of union of the graft and both fragments (*Fig.* 41). Further X-ray plates on May 14 showed more union by bone. The patient returned to his work as a labourer on May 15, and was able to carry on without discomfort.

*Case* 5.—J. M., aged 37, labourer. Admitted on Oct. 1, 1932, with a history of a fall on June 8, resulting in a fracture of the distal end of the radius, with anterior dislocation of the semilunar bone and fracture of the carpal scaphoid. The other injuries had been treated successfully, but there was non-union of the fracture of the scaphoid. There was limitation of all movements of the wrist, including rotation, with weakness of the grip, and partial anæsthesia of the ulnar nerve distribution in the hand. Some swelling was present over the lower end of the radius and carpal bones, with fairly marked tenderness over the dorsal and palmar surfaces of the scaphoid. X-rays at this time showed union of the fracture of the radius, normal position of the semilunar, and a non-union of a fracture through the waist of the scaphoid, with both fragments viable.

Operation was performed on Nov. 1 by Dr. W. E. Gallie. The fracture surfaces were cleared and freshened, and on account of the direction and position of the fracture, it was thought to be easier to cut a groove in the distal fragment, through the ligamentous area,

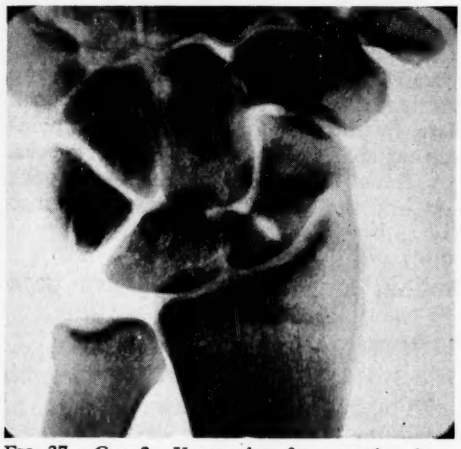

FIG. 37.—*Case* 2. X-ray taken four months after injury, showing non-union of scaphoid.

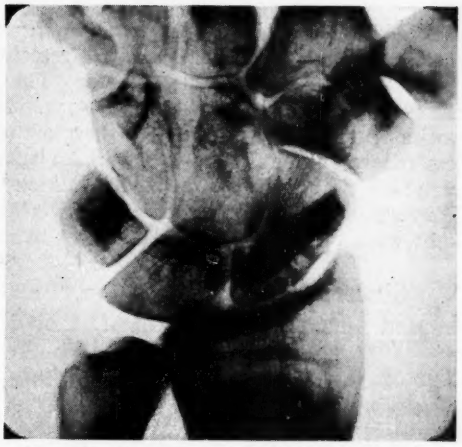

FIG. 38.—*Case* 2. X-ray taken after operation, showing the bone-graft in position.

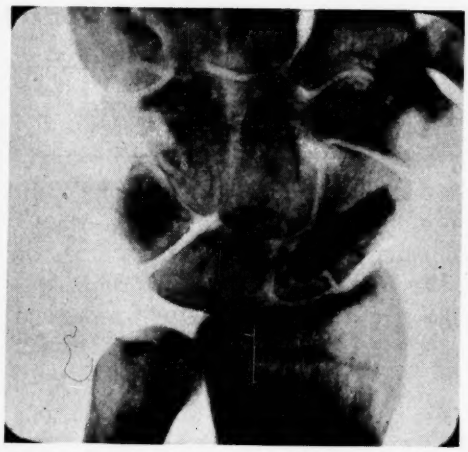

FIG. 39.—*Case* 2. X-ray taken four months after bone-graft, showing solid union of fragments.

FIG. 40.—*Case* 3. X-ray taken following operation, showing bone-graft in position.

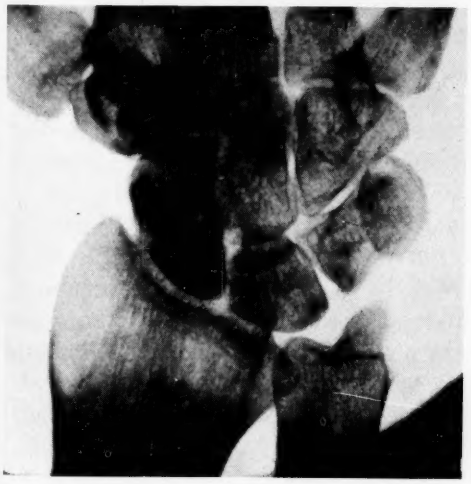

FIG. 41.—*Case* 4. X-ray taken eight weeks after operation, showing union of bone-graft of both fragments, and fracture line disappearing.

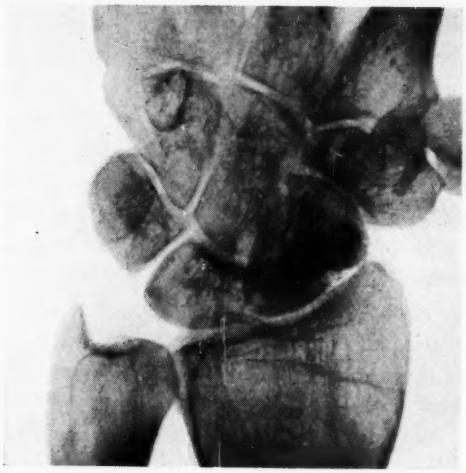

FIG. 42.—*Case* 5. X-ray taken twelve weeks after bone-graft, showing union of graft and fragments.

and to drill a hole in the proximal fragment. A graft from the tibia fitted nicely into both fragments, and the fracture surfaces remained in fairly good apposition.

A circular plaster was applied. The post-operative course was not complicated and the plaster was removed on Jan. 4, 1933. X-rays taken at that time showed the graft in good position, with signs of union of the graft to the distal fragment. There was free and painless movement of the wrist-joint. An anterior cock-up splint was applied for four weeks, at which time X-rays showed firm union of the fragments to each other and to the graft (*Fig.* 42). Movements of the wrist were very good, and the patient was able to return to light work on March 15, and to heavy work on May 1.

Since submitting this paper for publication, four other cases have been treated successfully by this method.

## CONCLUSIONS

1. Recent fractures of the carpal scaphoid may be difficult to see on X-ray films, but later are seen easily.

2. Many cases if treated early will unite by bone, but if neglected may develop non-union.

3. Non-union treated by bone-graft has given excellent results with complete recovery of function in our cases.

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# X-RAY INVESTIGATION OF THE UPPER RIGHT QUADRANT*

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With the exception of the introduction of the barium meal as an aid in diagnosis of lesions of the gastro-intestinal tract, no single contribution for the application of opaque media in radiological diagnosis has been as valuable as the work of Evarts A. Graham and his collaborators in developing and perfecting the use of sodium tetraiodophenolphthalein to demonstrate hepatic function and cholecystic disease.

One evening in the winter of 1922, Dr. Graham, who had given up his surgical work in 1913 and 1914 for the study of chemistry, was ruminating on the work of Abel and Rowntree which had demonstrated the almost complete excretion of chlorinated phenolphthalein through the bile. He conceived the idea that, by attaching atoms of some substance opaque to X rays to the phenolphthalein in place of the chlorine, it might be possible to obtain a shadow of the gall-bladder. Casting around for some such substance, he noted in a list of indicators supplied by the Eastman Company the free acid of tetraiodophenolphthalein. This indicator was obtained and Dr. Warren Cole converted it into the sodium salt on account of its greater solubility before injecting it intravenously into their experimental dogs. Six dogs were injected, and on the following morning when X-ray films were made five of the dogs showed no shadow in the gall-bladder, and one of them showed the gall-bladder faintly. On investigation it was found the animal keeper had forgotten to feed the dog in which the shadow of the gall-bladder was obtained and had fed the other five. If that keeper had been utterly efficient, we might not have had the art of cholecystography as it exists to-day!

From that time onward numerous other salts—forty-eight in all—were tried. Of these, thirteen gave gall-bladder shadows, but they all possessed disadvantages, and eventually, with better chemical preparation, the original salt of sodium tetraiodophenolphthalein was again utilized, and it is the basis of all the dyes used to-day, either orally or by intravenous injection.

Controversy for some time was rampant over the proper method of administration, whether the dye should be given by mouth or intravenously. Even to-day each side has its advocates, but the oral method has far out-distanced the intravenous one on account of its easier administration, slighter systemic reaction, and absence of danger of the serious results that may occur at the site of injection if given intravenously; and also in a well planned carefully carried out oral technique the percentage of correct diagnoses of cholelithiasis and cholecystitis is equal to that obtained by the intravenous method. During the last seven years we have used the oral method exclusively and find no reason to employ the intravenous.

* Read before the Section of Medicine, Academy of Medicine, Toronto, on Oct. 10, 1933.

## INDICATIONS AND CONTRA-INDICATIONS

The possibility of the presence of gall-bladder disease should be investigated in all patients complaining of pain or distress in the upper abdomen. For this investigation cholecystography should be used, but may we urge, and urge most strongly, that the gall-bladder investigation should not be the only examination? An opaque meal should always follow after cholecystography, and some cases will call for the administration of a barium enema, or the intravenous use of the colloidal solution of thorium dioxide, before a clean-cut diagnosis is obtained of the actual lesion present which simulates cholecystitis or cholelithiasis. The examinations should be carried out in the order named above. If the meal, enema, or thorium is administered first, the cholecystogram will be obscured by barium in the bowel or thorium in the liver.

Many conditions have been mentioned by other investigators as contra-indications to the administration of the dye. In our laboratory our experience has been that the only contra-indications are: chronic increasing jaundice, acute jaundice, myocardial degeneration, and advanced debility. In the chronic increasing jaundice we have never been able to fill the gall-bladder, and our line of investigation has been to rule out the presence of carcinoma of the pancreas, of the ampulla of Vater, or of the gall-bladder, by the barium meal or thorium dioxide; or the presence of a stone or obstruction of the common duct by flat films of the gall-bladder region. If the dye is administered in acute jaundice, serious damage to the liver cells may result, and several fatalities have been reported from such a procedure. When myocardial degeneration is present the systemic reaction from the exhibition of the dye may cause such a serious upset that a fatality may result. The same applies to the exhibition of the dye in advanced debility.

In secondary carcinoma of the liver where the primary is in the gastro-intestinal tract, in syphilis of the liver in its various forms, in hypertrophic cirrhosis, and in polycystic disease of the liver we have administered the dye with no ill effects, and have frequently been amazed at the presence of apparently normal function with a large amount of hepatic involvement present.

## METHOD OF ADMINISTRATION OF THE DYE

The oral administration of the dye calls for intelligent co-operation by the patient if accurate results are to be obtained. Often we found on inquiry that the lack of filling of the gall-bladder was due, not to disease, but to non-attention to some of the instructions given to the patient.

It is wise to have the colon empty twenty-four hours before the examination is carried out, castor oil or compound liquorice powder being the most suitable laxatives. The salines leave a large amount of gas in the colon which may overlie the gall-bladder region and simulate cholesterol stones with a calcium periphery.

On the evening of the administration of the dye the patient takes a large meal at 7 o'clock consisting of the following: bread, but no butter; vegetables, but no butter, oils, or fat; salads without oil; fresh fruit and jams—and only water to drink. At 8 o'clock the dye is taken and the patient immediately retires. The following morning we make the first examination at 9 o'clock with the patient fasting and only allowing enough time to dress and come to the laboratory. Even

the odour of food should be avoided, as we have been able to empty a normally filled gall-bladder by passing a freshly prepared dish of bacon and eggs under the patient's nose. Occasionally a gall-bladder is slow to fill, and if this occurs a second observation should be made in two hours, i.e., fifteen hours after the ingestion of the dye. Then a fat meal is given and the last examination made at eighteen hours after the ingestion of the dye. If the gall-bladder filled well at the first examination, the second one may be omitted, the fat meal taken at once, and the examination for emptying made at the eighteenth hour. The question is often asked: If vomiting occurs, does it render the examination useless? In our experience we have found that vomiting taking place thirty minutes after drinking the dye has no effect upon a satisfactory observation, because within that time sufficient of the sodium tetraiodophenolphthalein has passed to be absorbed within the intestinal tract and give a satisfactory concentration within the gall-bladder if it is normal. The same answer applies to the attacks of diarrhœa occasionally occurring with the exhibition of this salt. Unless the diarrhœa is extremely severe and manifests itself within a short time, it will have no effect upon the end-result. If dye is observed in the cæcum and ascending colon at the first observation, we conclude the patient has absorbed sufficient to show a well filled gall-bladder if this viscus is functioning normally.

The dye is absorbed from the digestive tract, excreted by the liver, passes to the gall-bladder, where it is concentrated by the mucosa along with the normal bile, and from there it passes through the cystic and common ducts again into the digestive tract.

It is not necessary to go into full detail of all the radiological technique—suffice it to say a kilovoltage as low as will completely penetrate the individual should be used, thus obtaining a soft film with a wealth of detail. The milliamperage will be governed entirely by the length of time needed for the exposure. Numerous films should be made, and the whole area from the 9th rib to the lower portion of the sacro-iliac joint should be covered if a thorough demonstration of the gall-bladder area is to be made.

## POSITION OF THE GALL-BLADDER

The gall-bladder follows the same rules of habitus as do the viscera so well demonstrated by the late Dr. Walter Mills, of St. Louis. He divided the types of habitus into four, and gave us the hypersthenic, sthenic, hyposthenic, and asthenic classifications. In the hypersthenic individual the gall-bladder is found high, level with the 10th to 12th ribs, well out from the spine and lying in an almost transverse position. In the sthenic and hyposthenic type it is placed more obliquely, situated at a lower level and closer to the spine, while in the asthenic individual it will be level with or below the iliac crest and close to or superimposed upon the spinal column.

## NORMAL FILLING

The normal gall-bladder commences to fill in eight hours, is well filled in thirteen, and the greatest concentration occurs about the sixteenth hour after ingestion. It should be empty three hours after the ingestion of a fat meal. In the sthenic to the asthenic type it is pear-shaped, and we divide it into the fundus,

body, and neck. In the hypersthenic it lacks this characteristic and is practically oval in appearance. The average content in all types is about one and a half fluid ounces. The mobility varies in the different types, is least in the hypersthenic and most mobile in the asthenic type. One must never forget the possibility of a situs transversus when the shadow is not seen on the right side, and inspection of the left side of the film should be made to rule out this possibility. Generally speaking, a gall-bladder that fills well at the first observation, shows contraction and increased concentration at the second examination, and is empty three hours after the fat meal, may be considered normal. (*Figs.* 43, 44.)

FIG. 43.—Normal gall-bladder, showing concentration of the dye at the first examination.

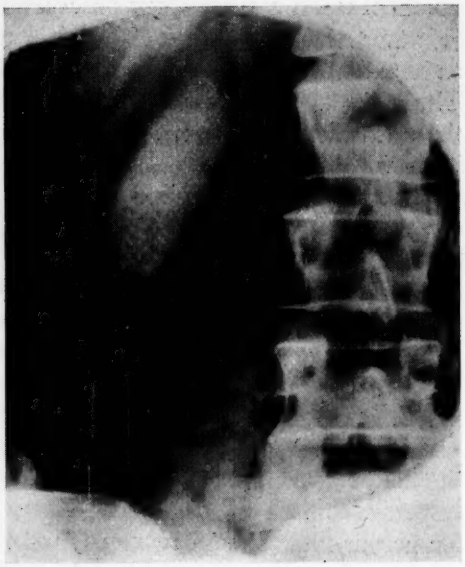

FIG. 44.—Normal function of gall-bladder containing cholesterol stones. These stones would probably be missed if the concentration of the dye had not occurred.

## ABNORMAL FILLING

While the gall-bladder may be filled well and empty well, the smooth pear- or oval-shape may show some deformity, and it is well to bear in mind that this may be due to extrinsic pressure before any statement of pericholecystic adhesions is made. Before the latter can be established, the deformity in contour should be present on the films of a subsequent examination, and, as in duodenal ulcer or carcinoma of the stomach, should show no change during the first or subsequent observations. In some cases a constant deformity in a well filled gall-bladder is due to a small ulcer of the mucosa accompanied by muscular contraction similar to the spasmodic incisura noted in gastric ulcer, or it may be present after such an ulcer has healed like the organized hour-glass deformity in the stomach (*Fig.* 45).

During the examination the gall-bladder may be fairly well filled, and at the subsequent examinations the viscus is slow to empty. In our opinion this points towards a chronic inflammatory change, the mucosa being damaged (*Fig.* 46) but its concentrating function not completely destroyed, and it is accompanied by some

FIG. 45.—Intrinsic scar due to chronic fibrotic change similar to organized hour-glass in the stomach.

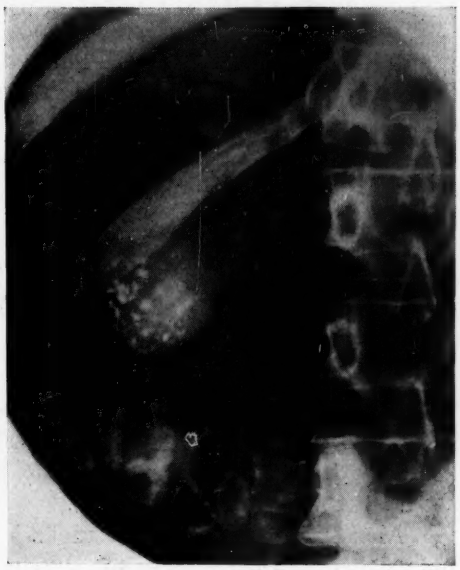

FIG. 46.—Slight damage to mucosa shown by poor concentration, and numerous stones present in the fundus.

FIG. 47.—Gall-bladder slightly dilated; function subnormal. Several gall-stones are shown, one in the cystic duct acting as a ball-valve allowing the gall-bladder to fill, but not allowing it to empty.

FIG. 48.—Pericholecystitis shown by adhesions to descending duodenum. Notice the absence of valvulæ conniventes.

fibrotic changes in the wall. If 40 per cent residue is present three hours after the administration of a fat meal, and the density is equal to that observed on the original films, we believe one is justified in a diagnosis of chronic cholecystitis.

When the gall-bladder fails to fill at any of the periods throughout the examination, while the probabilities are that the reason for this phenomenon is a well-marked cholecystitis, other lesions may be the cause, and the radiologist should make a diagnosis of mal-function, at the same time searching by the meal and enema for other lesions which may account for the non-filling. A stone situated in the hepatic, cystic, or common ducts may be the answer to the non-visualization (*Fig.* 47), and if sufficient calcium is present it will undoubtedly show upon the films.

Often an acute duodenal ulcer or a gastric ulcer close to the pylorus will give a non-filled gall-bladder, which at operation and under the pathologist's microscope is normal. Whether this is due to some communication between the special sympathetic branches where the ulcers are present and the sympathetic branches with which the gall-bladder is so richly supplied, we are unable to state at the present time. An acute appendix will often also give us the same non-filling in a normal gall-bladder, and other acute abdominal lesions we have found occasionally set up the same reflex.

Malignancy of the gall-bladder is demonstrated only by complete non-filling, unless there is extension to the duodenum, and then the deformity is noted by the meal. It is possible that sufficient healthy mucosa could remain in carcinoma of the gall-bladder to concentrate the dye and show an actual deformity as in carcinoma of the stomach when the barium meal is used, but in our experience we have never filled a gall-bladder when this type of pathology is present and we have had to satisfy ourselves with a diagnosis of mal-function. (*Fig.* 48.)

Non-visualization may occur for other reasons. An enlarged kidney, retroperitoneal tumour, the high fundus of the uterus in the later months of pregnancy, or a large intra-abdominal cyst may press upon the cystic duct and thus not allow a normal gall-bladder to be demonstrated. Carcinoma of the hepatic ducts and hepatitis may also be the cause of non-filling.

Hydrops of the gall-bladder (*Fig.* 49) may easily be mistaken for the shadow caused by the right kidney, as the viscus in its enlargement assumes a shape resembling the renal contour. The soft film will, however, show the renal shadow separate from the markedly distended gall-bladder filled with the poorly concentrated dye.

Biliary calculi (*Figs.* 50–52) have been classified according to their chemical composition. From an academic standpoint this is very desirable, but the task of the radiologist is much simplified and less confusion caused in the mind of the clinician if they are simply classified according to the calcium content present. Those rich in calcium are known as ' opaque ' to the ray, and those wholly lacking or possessing only a slight amount of calcium, such as cholesterol stones, as ' non-opaque '. The last-mentioned type will be missed on the film very often unless a fair degree of concentration of the tetraiodophenolphthalein has been obtained. Fortunately with these types of stones a large percentage of the cases show slight damage to the mucosa, and the gall-bladder is seen well filled with the concentrated salt, the stones appearing as small dark shadows in the white gall-bladder. Papillomata of the gall-bladder may simulate these stones. The differential diagnosis

FIG. 49.—Hydrops of the gall-bladder. The markedly distended gall-bladder is shown pressing upon the barium-filled transverse colon.

FIG. 50.—Cholesterol stones in deformed gall-bladder due to pericholecystitis, and cystic duct dilated from obstruction.

FIG. 51.—Cholecystitis as shown by non-filling of the gall-bladder. Numerous faceted stones present.

FIG. 52.—Pericholecystitis shown by deformed gall-bladder. Function of gall-bladder subnormal. Cholesterol stones are present.

lies in the fact that on the various films stones will move about and assume different groupings in the fundus, body, or neck. This is particularly well seen during the period of concentration and contraction of the viscus. With papillomata the grouping will always be the same; only a closer approachment of the individual shadows is noted as the serial films are made. Another frequent source of error or confusion with this type of stone is gas in the second portion of the duodenum. The fenestration of the valvulæ conniventes duodeni simulates stones or papillomata very closely, but on precise observation of the various films these shadows will be noticed partially within and partially without the periphery of the filled gall-bladder, thus definitely locating them within the duodenum. A polypus of the gall-bladder may simulate a non-opaque stone, but rarely does a stone of this type attain the size of the usual polypus without sufficient calcium content present to show a well-marked periphery. The opaque stones may be well visualized in a gall-bladder still capable of carrying on its concentrating function, the varying amount of calcium content showing denser than the dye-impregnated bile. From this we see that the presence of either type of stone may be demonstrated in a gall-bladder apparently functioning normally.

For a considerable time we were of the opinion that a gall-bladder possessing stones but carrying on a normal function might well be treated by watchful waiting. However, considering the high incidence of carcinoma with an early history of gall-stones we now feel that the demonstration of cholelithiasis should be accepted as an indication for surgery.

When a gall-bladder shows no filling and shadows suggesting gall-stones are present, one must always remember that many other calcified deposits in this region may simulate on the film the presence of stone, and one must be on guard that an erroneous diagnosis of gall-stones is not added to the correct one of mal-function as shown by the non-filled gall-bladder. Renal calculi, calcification of Glisson's capsule, calcified lymph-glands, calcification of the suprarenal (*Fig.* 53), calcified rib cartilages, calcareous degeneration of the right renal artery, pancreatic calculi, dermoid cysts (*Fig.* 54), moles on the skin, fæcal concretions in colonic diverticula, all cause shadows simulating gall-stones, and each must be carefully differentiated.

A small percentage of gall-stones with cholecystitis perforate, establishing fistulæ into the adjacent viscera. We have seen these tracts established into the pylorus, the duodenum, and the colon (*Fig.* 55). Unless the barium meal and enema are added to the investigation, only a partially correct conclusion is reached by the demonstration of mal-function, and the surgeon encounters a much more difficult operation than would be the case had the radiologist carefully prepared his work beforehand. By the use of the meal or enema, the fistula is well seen and the gall-bladder filled with the barium. Occasionally after such a perforation has occurred into the colon the first clinical manifestation points towards colonic obstruction; for this reason the radiologist should be on the lookout for a large stone at the site of obstruction. Gall-stones may be found at any point within the colon; in one case referred as subacute appendicitis we observed a perforated gall-bladder, the stones had dropped into the cæcum, and at our examination we found three gall-stones lodged in the appendix.

Ulcers of the pylorus and duodenum (*Fig.* 56) may perforate into the common duct, setting up cholecystic symptoms, but with the barium meal the common and

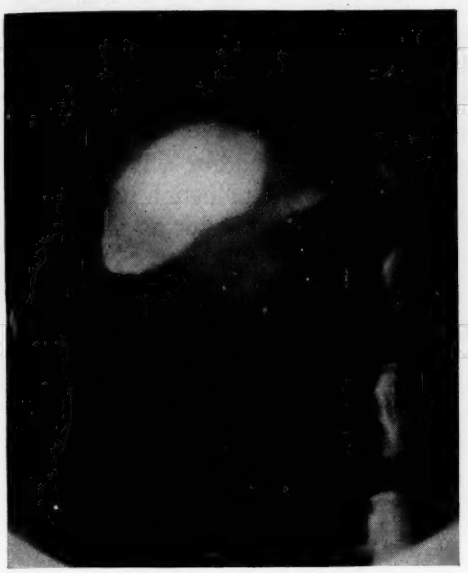

FIG. 53.—Cholecystitis demonstrated by non-filling. Calcareous shadow is due to calcification of the right suprarenal.

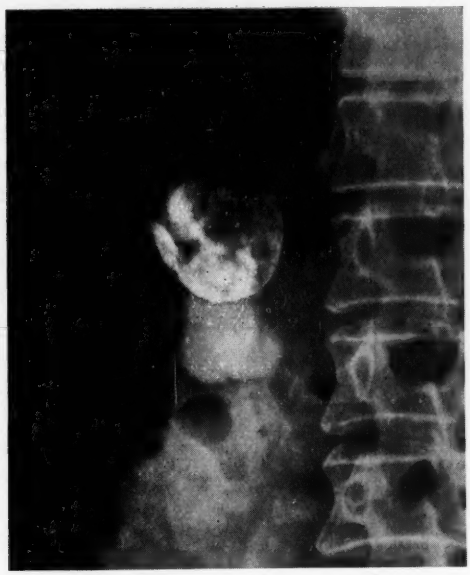

FIG. 54.—Gall-bladder carrying on normal function, but a double fundus is seen. Shadow above the gall-bladder is due to a dermoid cyst. Notice the shadow of bone fragments and of teeth.

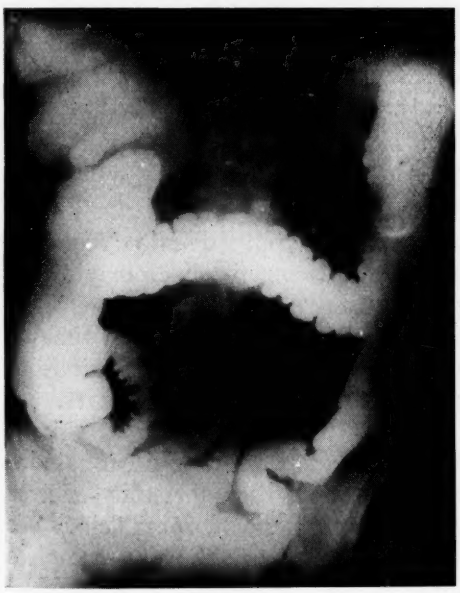

FIG. 55.—Fistula established between gall-bladder and colon following ulceration. Gall-bladder filled during flow of enema.

FIG. 56.—Perforated duodenal ulcer. Fistula established into common bile-duct. Notice common duct and hepatic ducts filled with barium.

cystic ducts and the hepatic tree will be visualized, showing the primary lesion to be gastro-intestinal and that of the gall-bladder secondary.

Frequently the first lead given towards gall-bladder disease is found in deformity of the pylorus and cap simulating either pyloric ulcer, early gastric carcinoma, or duodenal ulcer. However, this deformity is not as constant as in any of these conditions, and is caused by a veil or membrane, the so-called Morris membrane, extending from the gall-bladder and due to pericholecystitis. If the gall-bladder fills with the function test, deformity in its contour will be noted owing to these adhesions, and on fluoroscopic examination and palpation its mobility will be much lessened. In the normal stomach and duodenum during the screen examination and upon the films, the duodenal caput should ride true upon the pylorus, with the sphincter placed centrally, and angulation of the duodenal bulb or inconstant deformity should make one suspect the presence of pericholecystitis. Pericholecystic adhesions may also extend to the second portion of the duodenum. This part of the first loop of small intestines may be angulated, lacking in fenestration owing to tension from the adhesions, or present a convex curve due to pressure from the dilated gall-bladder. The adhesions may also extend to the colon, and in the proximal part of the transverse colon a pseudo-hepatic flexure is formed.

Pylorospasm is a frequent indication of gall-bladder disease during the fluoroscopic examination and upon the films. The pyloric canal—and by this we mean the portion occupied by the fan-muscle—never contracts or expands in a normal manner. We suspect, but at this time have not absolute proof, that the reason of this is some communication between the special branch of Opitz controlling the fan-muscle and the sympathetic supply of the gall-bladder.

## THORIUM DIOXIDE IN GALL-BLADDER INVESTIGATION

When the dye has been concentrated in the gall-bladder and the position of the viscus is in marked variation with the habitus of the patient, it is probably due to some enlargement or displacement of the liver. Radiological examination by the ordinary technique will be of little assistance, but the administration of colloidal thorium dioxide intravenously will give a well-visualized liver, and a detailed study of structural changes may be made. Cirrhosis, both the hypertrophic and atrophic type, syphilis, hydatid cysts, polycystic disease (*Fig.* 57), secondary carcinoma, and retroperitoneal tumours displacing the liver may all be shown. We have now used this solution for more than two years, and as yet have seen no ill effects, nor have any of our experimental animals killed from time to time shown any histological changes which could be interpreted as injurious to the liver or spleen, where 80 per cent of the retention of this medium occurs.

Not only as an indicator of gall-bladder function may the sodium tetraiodophenolphthalein be used; it is indeed valuable also as a basis for study of liver function, similar to the tests of Rosenthal with phenoltetrachlorphthalein. We beleive that patients with a fairly definite gall-bladder history showing complete retention of the dye will be much better surgical risks if, instead of being submitted to immediate operation, they are placed at rest in bed on an abundant carbohydrate diet in the form of glucose, some calcium, and large amounts of

water, and re-examined radiologically at a later period. In these cases we have found at the second examination concentration of the dye present, the complete retention having passed, and surgery may be undertaken with the expectation of a lower fatality rate.

Occasionally cases of intestinal allergy will give symptoms identical with those of gall-bladder disease. If these cases are investigated with cholecystography and

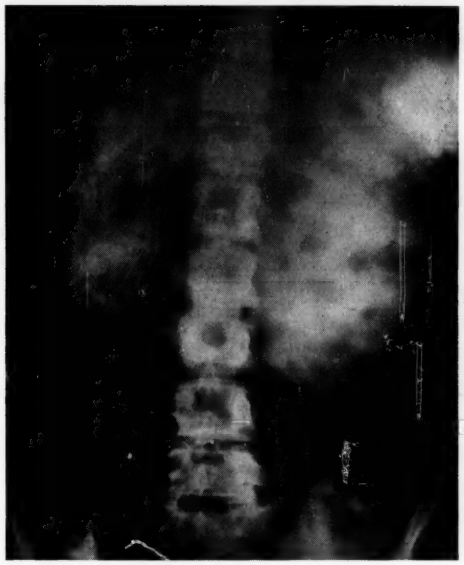

FIG. 57.—Polycystic disease of the liver shown by thorium dioxide. This case was referred as probable carcinomatous secondaries in the liver.

the ordinary opaque meal and found normal, we would suggest a series of sensitization tests before a definite medical or surgical régime is established.

In conclusion, may we plead for the closest collaboration between the physician, the surgeon, the pathologist, and the radiologist? If this is brought about, we shall not only increase the number of correct diagnoses, but also lower the mortality rate from disease in the upper right quadrant.

# CHANGES IN THE BLOOD-SUGAR LEVEL ASSOCIATED WITH SURGICAL OPERATIONS

By A. G. WEDDELL and H. E. D. GALE, London

The work which follows was done in the Surgical Professorial Unit of St. Bartholomew's Hospital at the instigation of its Director, Professor Gask.

It is a fairly general routine procedure to administer glucose by the rectum to patients who have undergone a severe surgical operation. Recently doubt has been thrown on the wisdom and usefulness of this procedure by Levi.[1] His observations led him to conclude that glucose rectal salines are not only useless but even harmful. Indeed, he proposed that in cases of post-operative shock it would be more in the interests of the patient to administer insulin.

The level of the circulating blood-sugar is a balance between income and expenditure. Ingested carbohydrates are absorbed from the alimentary tract in the form of glucose and then carried to the liver, where they are converted into, and stored as, glycogen. As required, this glycogen is reconverted into glucose and passes into the blood-stream, maintaining the blood-sugar level within certain limits, i.e., 80 to 120 mgrm. per cent. This represents a physiological cycle: Portal sugar —→ liver glycogen —→ circulating sugar. This cycle is influenced by nervous hormonic factors. The sympathetic-adrenalin mechanism promotes the change of glycogen into sugar, the vagal-insulin the conversion of sugar to glycogen and perhaps the utilization of sugar by the tissues. Anything tending to augment the sympathetic-adrenalin mechanism, such as activity, an increased metabolic rate as found in thyreotoxæmia, emotion, anæsthesia, restlessness, pain, or trauma, produces more adrenalin, thereby raising the level of the blood-sugar. On the other hand, food, rest, sleep, tranquillity, and freedom from pain augment the vagal-insulin mechanism, tending to produce a lowering of the level of the blood-sugar. Thus from general principles we should expect that emotionally stable patients, who are in good condition before the operation and whose convalescence is uninterrupted by pain or other adverse factors, should have a lower blood-sugar level than patients in whom these conditions are reversed. In other words, we should expect the level of the blood-sugar to run parallel with the clinical condition of the patient.

In view of the above criticism of the post-operative treatment of surgical cases it is of importance that the changes in the blood-sugar level as affected by surgical operations should be re-investigated. In the course of such an investigation it is necessary to consider first of all the effect of the anæsthetic. It is well known that the amount of sugar in the blood rises during anæsthesia. That this is due to the liberation of adrenalin is known, and recently Reid and Banerji[2] have shown that the actual sugar level in animals depends quantitatively on the amount of adrenal medulla available. In addition to the influence of the anæsthetic it is necessary to estimate if possible the extent, duration, and variation in the blood-sugar level which

may occur as the result of the operation itself. With such knowledge accurately obtained it might then be possible to assess the need or otherwise for glucose rectal injections and perhaps judge if other procedures might be useful.

## METHOD OF INVESTIGATION

The present investigation was confined to patients undergoing major operations. In order that the results obtained from all patients might be as parallel as possible, the blood was collected at the following periods: (1) On the day previous to the operation after a fast of twelve hours—this gave a resting level; (2) Half an hour before the operation; (3) After the induction of surgical anæsthesia when the skin incision was being made; (4) At various stages throughout the operation, these corresponding as far as possible in all cases; (5) Immediately after the operation when the patient had been returned to bed; (6) Six hours after this; (7) On the following morning, approximately twenty-four hours after the operation.

Blood was obtained from the finger except during the operation, when it was taken from the great toe because this was less disturbing to those taking part in the operation. The same puncture usually sufficed for all the specimens collected during the operation. The time at which the samples were taken was accurately noted for the sake of producing graphs (*Figs.* 58–66) which could be easily compared.

The estimation of the blood-sugar was by a modified Hagedorn and Jensen's method as used in this hospital by Dr. G. Graham.[3] By this method only small quantities of glutathione, the other main reducing substance in the blood, are included in the estimation. We observed the following special precautions in making the estimations. The blood was accurately measured in a narrow-bore pipette designed to deliver 0·1 c.c. All filter papers were, of course, free from starch. We avoided handling these as far as possible, since this is a known way of contaminating them with reducing substances. The papers were wetted with distilled water before filtering the precipitated proteins to prevent the loss of glucose during washing. We found that extreme care was necessary in the measurement of the known amount of potassium ferricyanide, an error of one drop affecting the reading to the extent of 10 mgrm. per cent. The titrations against thiosulphate were done with a microburette in order to obtain an accurate end point. Two samples of blood were collected for each estimation, the mean between the two being taken as the reading. Ordinarily a difference of 10 mgrm. per cent between the two estimations is considered good. By strict attention to the details of the technique the differences in our readings were within 5 mgrm. per cent, and in most cases not more than 1 or 2 mgrm per cent. All results with a difference of above 5 mgrm. per cent were discarded. The thiosulphate solution was freshly made for each series of estimations.

## CONCLUSIONS DRAWN FROM THE GRAPHS

**Pre-operative Observations.**—Examination of the curves obtained shows that the blood-sugar level in patients about to undergo a major operation does not vary much from the resting level. It is known that persons who are in a state of anxiety or apprehension have a raised blood-sugar level. It might perhaps be

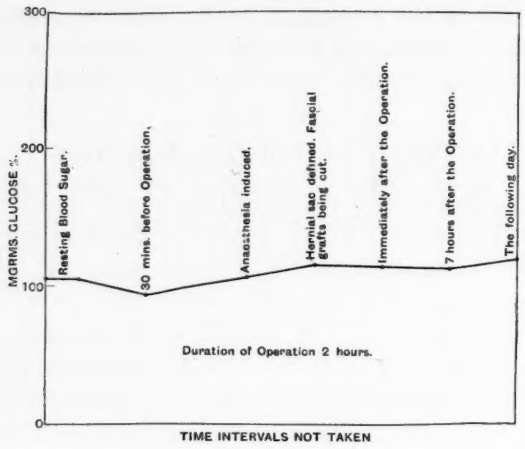


FIG. 58.

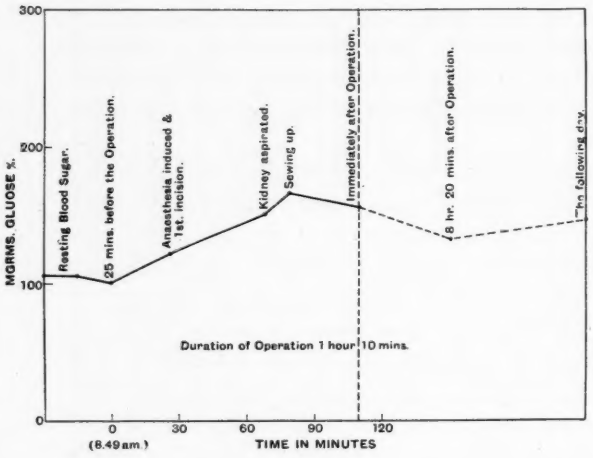


FIG. 59.

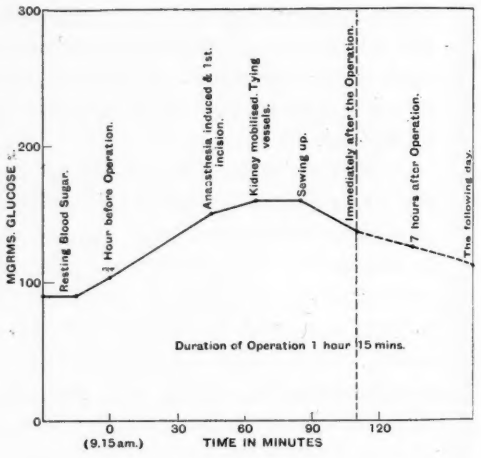


FIG. 60.

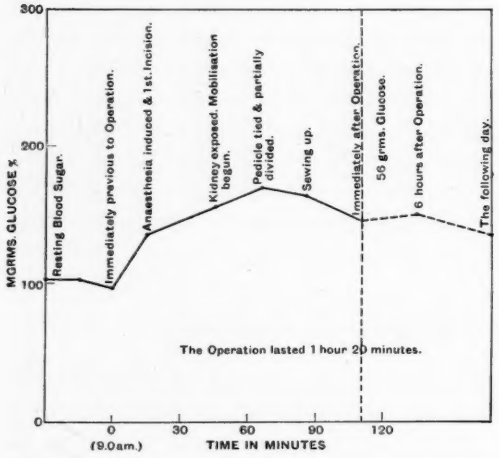


FIG. 61.

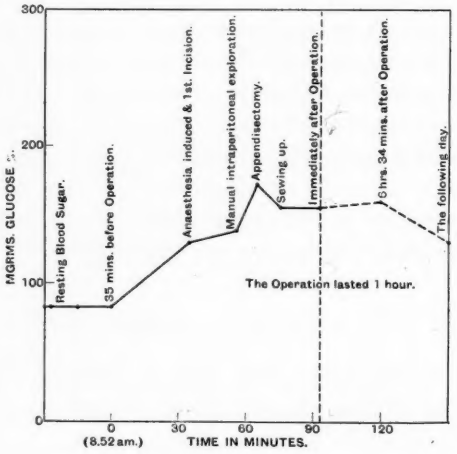


FIG. 62.

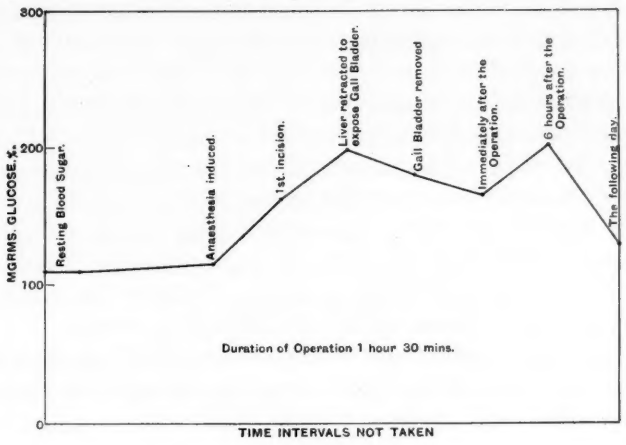


FIG. 63.

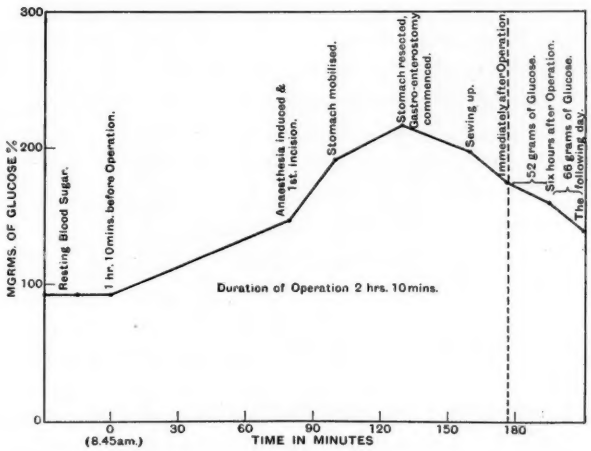


FIG. 64.

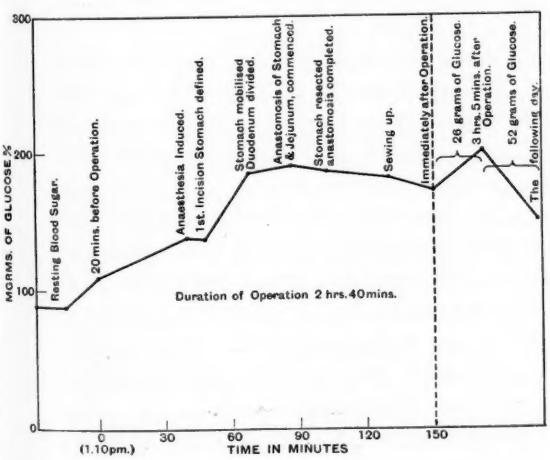


FIG. 65.

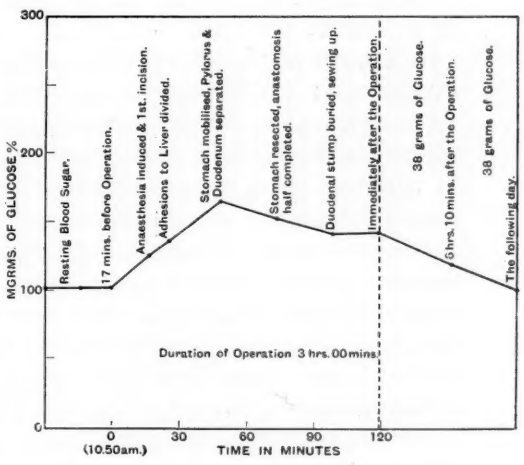


FIG. 66.

surmised from this that the ordinary hospital patient is not apprehensive or even greatly concerned about his approaching operation. We do not know whether this is a tribute to surgical skill or to the mental capacity of the patients.

**Effect of Anæsthesia.**—Induction of surgical anæsthesia has produced an increase in the blood-sugar level of variable extent. The average lies between 20 and 50 mgrm. per cent. The anæsthetic used in all cases was gas, oxygen, and ether. Induction of anæsthesia caused a larger average rise in males (36·5 mgrm. per cent) than in females (23 mgrm. per cent). The number of female cases was, however, only three. This suggests, so far as it goes, that the threshold of adrenalin release may be higher in females than in males. Perhaps this is another indication of the greater degree of stoicism usually attributed to women.

Further comparison of the graphs brings out the fact that the maximum height of the sugar level above its resting value is not correlated with the duration of the anæsthesia.

*Table I.*—Relation between Duration of Operation and Rise of Blood-sugar Level.

| | Mrgm. per cent above Resting Level | Duration |
|---|---|---|
| | 124 | 2 hr. 10 min. |
| | 101 | 2 hr. 40 min. |
| (*a*) | 88 | 1 hr. 00 min. |
| | 88 | 1 hr. 30 min. |
| | 70 | 1 hr. 15 min. |
| | 67 | 1 hr. 20 min. |
| (*b*) | 63 | 3 hr. 00 min. |
| | 62 | 1 hr. 10 min. |
| | 10 | 2 hr. 00 min. |

The relationship between the maximum rise and the duration of the operation is set out in *Table I.* Taking the two extreme cases (*a* and *b*) to illustrate this point, it will be seen that in the operation lasting one hour there was a rise of 88 mgrm. per cent, while in the other lasting three hours there was only a rise of 63 mgrm. per cent.

**Nature of the Operation.**—The nine cases investigated can be divided into two groups: (1) Intraperitoneal; and (2) Extraperitoneal. In *Group* 1 (3 partial gastrectomies, 1 cholecystectomy, and 1 exploratory laparotomy followed by appendicectomy) the average maximum rise was 93 mgrm. per cent, while in *Group* 2 (2 nephrectomies, 1 nephrostomy, and 1 ventral hernia repaired with fascial grafts) the average maximum rise was 52 mgrm. per cent. From this it seems that operations which take place nearer to the splanchnic area, i.e., in the vicinity of the solar plexus, cause a greater rise in the blood-sugar level than those placed at a distance from this point. It is interesting to note in *Fig.* 63 that the maximum blood-sugar level was reached while the liver was being palpated. This relation is even better shown in *Fig.* 62, where, ten minutes after the manual exploration of the abdominal viscera, there was a peak in the blood-sugar level.

**Post-operative Observations.**—In patients who appeared comfortable and relatively free from pain on the following day, the blood-sugar was found to have

reached a lower level than in those who were uncomfortable and in pain. This is shown in *Table II*, where short clinical notes and the chief points of moment in the graphs are tabulated.

Four patients were given glucose rectal salines, which were retained, and the blood-sugar level was estimated on the following day. Of these four cases the blood-sugar level had already returned to normal in one case and in the other three it was falling. It was therefore concluded that these patients were utilizing the glucose administered by the enemata.

## DISCUSSION

From the evidence produced it appears that the hypothesis made earlier, that the blood-sugar level runs parallel with the clinical condition of the patient, is justified. Operations on the upper abdomen around the cœliac ganglia are more marked in their effect on the blood-sugar level than those placed more remote from this region, irrespective of their duration. If a high blood-sugar level is to be avoided gentle handling is an essential factor in this neighbourhood.

It has been claimed that in a series of normal and diabetic patients the administration of 50 to 80 grm. of glucose in 500 c.c. of saline occasioned either a fall or at the most only a slight rise in the blood-sugar level. It was concluded therefore that sugar was not absorbed from the colon, in contrast to its absorption from the stomach, this leading to a pronounced change in the blood-sugar level. In our series the enemata were always retained and the bowels remained unmoved for twenty-four hours. So it seems reasonable to us to conclude that absorption had taken place. The effect on the blood-sugar level will depend on several factors, the rate of absorption, the rate at which the sugar is being produced or used up. The problem of absorption cannot be resolved by direct appeal to the blood-sugar level at any one moment. Absorption of glucose from the rectum is at the rate of 6 grm. per hour (Starling) or 3 grm. per half hour: 50 grm. of glucose given by mouth produces a maximum rise of about 60 mgrm. per cent in the blood-sugar level in half an hour. Assuming all factors to be constant, 3 grm. would only give a rise of 4 mgrm. per cent. Thus no great rise can possibly be expected to occur when glucose is given per rectum. Further detailed work on this problem has been done by Collens and Boas,[4] who produced additional evidence for the absorption of glucose from the rectum. In all our cases the blood-sugar level, although above normal, continued, after no rise or a very slight one, to fall despite glucose enemata. From our observations we have no evidence that glucose enemata are harmful or irritant. Rather it would seem that their use is justified on the ground that they replace the depleted sugar stores of the body.

The raised blood-sugar level shown in these curves must depend to some extent upon a diminished utilization of glucose by the tissues, since the patients are in a depressed condition during and for some time after an operation. It has been suggested that insulin would be of value presuming that it would increase utilization. The carbohydrate stores must have been depleted to some extent during the operation, and an injection of insulin would still further diminish these stores unless it was combined with glucose. In any case, if the area beneath a graph made from the curve in *Fig.* 64 (extended to cover a twenty-four hour period) is calculated, the amount of glucose represented is 27 grm., which is

*Table II.*—Changes in the Blood-sugar Level Associated with Surgical Operations

(Nine Cases)

| Case No. | Age and Sex | Operation | Duration | Glucose in Mgrm. per cent | | | | | Rectal Glucose | Condition | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Resting Blood-sugar | Pre-operative Rise or Fall | Rise after Induction of Anæsthesia | Maximum Rise above Resting Level | B.S. Twenty-four hours after Operation | | Pre-operative | Post-operative |
| | | | Hrs. Mins. | | | | | | Grm. | | |
| 1 | M. 61 | Hernia .. .. | 2 00 | 105 | −11 | 1 | 10 | 16 | 0 | Good | Little pain |
| 2 | F. 28 | Nephrostomy | 1 10 | 105 | − 6 | 16 | 62 | 40 | 0 | Pyrexial | Some pain |
| 3 | M. 40 | Nephrectomy | 1 15 | 90 | +13 | 60 | 70 | 22 | 0 | Fair | Little pain |
| 4 | M. 62 | Nephrectomy | 1 20 | 102 | − 5 | 33 | 67 | 33 | 56 | Fair | Cough |
| 5 | F. 28 | Laparotomy | 1 00 | 83 | 0 | 47 | 88 | 47 | 0 | Good | Vomiting |
| 6 | F. 40 | Cholecystectomy | 1 30 | 110 | — | 6 | 88 | 20 | 0 | Good | Good |
| 7 | M. 46 | Partial gastrectomy | 2 10 | 93 | 0 | 53 | 124 | 47 | 118 | Fair | Some pain |
| 8 | M. 64 | Partial gastrectomy | 2 40 | 89 | +21 | 50 | 101 | 61* | 78 | Hb per cent low. Poor | Weak and feeble |
| 9 | M. 49 | Partial gastrectomy | 3 00 | 103 | 0 | 22 | 63 | 0 | 76 | Good | Very good |

* Eighteen hours after operation in this case.

equivalent to 108 calories. The basal metabolic requirements may be taken as 1800 calories. Thus it will be seen that the amount of excess of sugar circulating in the blood is small. The case on which this calculation is based showed the highest maximum rise in blood-sugar level. Now in *Fig.* 58 this area is negligible, so that it will be seen that the routine administration of insulin and glucose as a part of post-operative treatment cannot be expected to have any specific beneficial effect.

From the cases which are the basis of this report we are inclined to believe that a persistently high blood-sugar level is a feature to be expected when the patient is not doing well. Such an expectation, if confirmed, would perhaps give some point to the suggestion that insulin combined with glucose might be of some service.

## SUMMARY

1. Pre-operative anxiety, as measured by the change in level of the blood-sugar, must be very slight.

2. The duration of the anæsthesia does not determine the height of the maximum rise.

3. There is a rise in the blood-sugar level during major operations, particularly marked when the splanchnic area is involved.

4. The rise in the amount of the blood-sugar level depends far more on the nature of the operation than on its duration.

5. We find no evidence that glucose rectal salines as a routine post-operative procedure have any specific effect. There is no reason to suppose that they are either useless or harmful, and they may be of value in that they form a convenient method of post-operative feeding.

6. The use of glucose and insulin in post-operative treatment is discussed.

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# DIVERTICULA OF THE VERMIFORM APPENDIX*

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THE first attempt at a comprehensive survey of diverticula of the vermiform appendix appears to be that of Herb in 1907. Further papers appeared in 1911 (MacCarty and McGrath), 1919 (Moschowitz), 1921 (Wilkie), 1923 (Stout), 1927 (Chase), and 1928 (Pack and Scharnagel).

During the five-year period 1927–31, 1493 appendices have been removed at operation at King's College Hospital. Of these, 8 showed the presence of diverticula. The percentage incidence represented by these figures is, therefore, 0·53 per cent. These figures, however, must almost certainly exaggerate the incidence, for in quite a large number of cases the normal appendix has been removed when the abdomen was opened for some other surgical condition and the fact not made sufficiently prominent in the records to enable one to trace it. The total number of appendicectomies therefore exceeds the above figure. Even when an allowance is made for this fact, the incidence is undoubtedly higher than the scant reference to the subject in the literature leads one to imagine. No single case has been observed in the last 2680 consecutive post-mortems. Diverticula of the appendix are easily seen, and it is unlikely that the examiner would miss them if present. The explanation of this striking fact is probably that diverticula of the appendix are almost invariably associated with inflammation of the appendix, and this leads to operative removal.

In none of 4631 barium meals and 695 barium enemas were diverticula of the appendix revealed by X rays. For the two cases of diverticulosis of the appendix diagnosed by X rays the author is indebted to colleagues. These cases are of considerable interest, for the appendix shares in a general diverticulosis of the whole colon. As will be seen, in the majority of cases in the author's pathological specimens there is an obstruction to the lumen of the appendix near the cæcal end. Such an obstruction will prevent the barium from entering the appendix, so that X rays will fail to reveal the diverticula. Thus in diverticulosis of the appendix we find an exception to the general rule that the diagnosis of intestinal diverticula depends upon X-ray examination.

The only figures of incidence which the literature offers are those of MacCarty and McGrath, who found 17 cases of diverticula in 5000 specimens (0·34 per cent), and those of Moschowitz, who found 4 cases in 1500 examinations (0·26 per cent).

**Age and Sex Incidence.**—Diverticulosis of the appendix shows no parallel in respect of age incidence with diverticula elsewhere in the intestine. The author's 12 cases (including the X-ray cases) show a wide range, the youngest

*A section of the Jacksonian Prize Essay, 1932.

being 18 and the oldest 64. The average age is only 42. Six were men and 6 women. The cases are as follows:—

| Specimen | Case No. | Age | Sex | Duration of Symptoms |
|---|---|---|---|---|
| 1 | 198 | 18 | F. | 48 hours |
| 2 | 199 | 64 | M. | 2 months |
| 3 | 200 | 41 | F. | 12 months |
| 4 | 201 | 48 | M. | 7 years |
| 5 | 202 | 27 | M. | 2 years |
| 6 | 203 | 30 | F. | 48 hours (?) |
| 7 | 204 | 28 | F. | 48 hours |
| 8 | 205 | 21 | M. | 2 years |
| 9 | 206 | 48 | F. | 6 years |
| 10 | 207 | 55 | M. | 5 years |
| 11* | 168 | 58 | M. | No symptoms due to appendix |
| 12* | 209 | 62 | F. | No symptoms due to appendix |

* Diagnosed by X rays.

In MacCarty and McGrath's series of 17 cases, by a curious coincidence, the youngest and oldest patients were the same ages as the author's, viz., 18 years and 64 years respectively. The average age at operation was 34 years, and the average duration of symptoms 7 years.

## DESCRIPTION OF SPECIMENS

In all cases the diverticula were multiple. Three were associated with acute, and seven with chronic, appendicitis.

Fig. 67.—*Case* 198. Diverticulitis of the appendix. (*Miss Barclay-Smith.*)

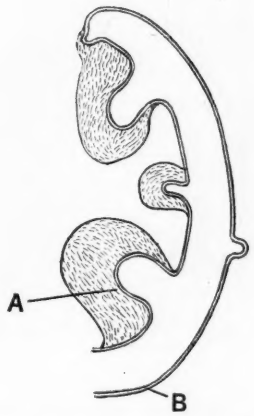


Fig. 68.—*Case* 198. Diagrammatic representation of the specimen illustrated in *Fig.* 67. (*Miss Barclay-Smith.*)

**Specimen 1** (*Case* 198).—The specimen (*Fig.* 67) was removed at operation from a girl of 18 who suffered from symptoms of acute appendicitis. There was

no previous history of pain. It shows an enlarged appendix cut through from the convex border, and opened out. The incision has also divided the meso-appendix. The whole of the appendix shows evidence of severe acute inflammation, being almost gangrenous in places. The wall is greatly thickened, and the mucous membrane dark from extreme congestion. Microscopic section shows an extensive infiltration of all the coats of the appendix with blood. The mucous membrane preserves the normal rugose appearance, and there is thus no evidence of persistent high pressure inside the lumen. The meso-appendix is thickened to many times the normal size. Pushing into it from the concave aspect of the appendix are four pouches, which have been bisected. The first and third of these (starting from the cæcal end) are of very large size. The second can only be seen on one half of the specimen, and the fourth is small, and near the tip. The line of section passes through a fifth diverticulum, situated on the convex border, $\frac{1}{10}$ in. from the cæcal end.

The first, second, and third diverticula form large, firm, rounded swellings, bulging into the meso-appendix, and can be clearly seen on the reverse side of the specimen. There is a cleft between the first and second pouches (*Fig.* 67.)

The wall of each of the first three diverticula is very thick, that of the first one measuring fully $\frac{1}{2}$ in. across. The position and relative thickness of the diverticula are best seen in the diagrammatic representation in *Fig.* 68. There is no obstruction to the opening from the appendix into the cæcum.

A section for microscopic examination was taken from **A** and **B**. **A** shows the wall of the first diverticulum and **B** a portion of the wall of the appendix itself. The whole of the diverticular wall is infiltrated with blood, a condition of hæmorrhagic infarction. The blood-cells lie in a framework of œdematous fibrous tissue, which represents the altered submucous coat. No muscle tissue is seen in the wall of the diverticulum, but is present in the section of the appendix wall, lying beneath a markedly thickened submucosa, which is infiltrated with blood.

SUMMARY.—An acutely inflamed appendix, showing hæmorrhagic infarction, bears five diverticula. Four pass into the meso-appendix and one from the convex surface. The wall of the diverticula contains no muscle.

FIG. 69.—*Case* 199. Diverticula of the appendix. The cæcal end of the lumen is obstructed, and the distal third obliterated. (*Miss Barclay-Smith.*)

**Specimen 2** (*Case* 199).—The specimen (*Fig.* 69) was removed at operation from a male patient aged 64 years, who had suffered from symptoms of chronic appendicitis for two months. The specimen has been divided along its long axis and opened to display the lumen. The appendix is shorter than the usual, and $\frac{3}{4}$ in. from the tip is kinked to a right angle. There is a deposit of fat in the meso-appendix at the angle thus formed. The lumen distal to the kink is completely obliterated by fibrous tissue, and the lumen at the cæcal end is markedly stenosed. Between these two obliterated portions, the lumen of the appendix is widely distended, and its wall is comparatively thin. The mucous

membrane is smooth. Opening out from this distended portion are some small diverticula. These are wide-mouthed, and have an extremely thin wall. If held up to the light, the wall looks like thin tissue paper, having no muscular coat. The fate of the muscle coat is clearly shown on naked-eye examination. If the convex margin be traced from the cæcal end to the tip, a small wide pouch is seen. At the mouth of this pouch there is a wide gap in the muscle coat. This occurrence is also shown an inch further along the border. Except for the gaps, the muscle coat of the ballooned part of the appendix is well developed, and shows a definite hypertrophy near the centre of this portion. Apart from the obliterative process at either end of the appendix, there is little macroscopic evidence of chronic inflammation. The diverticula themselves are quite free.

No microscopic section of this specimen was made. One needs to take a section completely across the appendix and a diverticulum to be of value, and such, unfortunately, would destroy the specimen. Other specimens (Nos. 9 and 10) were sacrificed to this end.

SUMMARY.—In this case the lumen of the appendix is stenosed at both ends. As a result of the proximal obstruction, the middle part is ballooned out. The wall is thin, but the muscle coat is slightly hypertrophied. There are gaps in it, through which the mucous membrane has herniated to form diverticula. These have very thin walls. The situation of the diverticula is not regular. There is one on the antimesenteric aspect, and the two largest are situated on the surface between the concave and convex aspects.

FIG. 70.—*Case* 200. Diverticula of the appendix. The muscular coat is well defined, except in the proximal half on the convex aspect. At A a portion has been removed for section.

**Specimen 3** (*Case* 200).—The specimen (*Fig.* 70) was removed at operation from a woman aged 41 who had suffered from attacks of pain in the right side of the abdomen for a little over twelve months. The specimen has been split longitudinally from the convex surface, and shows a long and thickened appendix. The lumen near the cæcal end is very narrow, but not completely obstructed. A further narrowing occurs about half way along. There is no distension of the lumen in any situation. The mucous membrane is very much thickened throughout, and the lumen is not easily traced. Small bristles have been placed in the mouths of pockets formed by the mucous membrane pushing through the muscle coat. Most of these are small, but on the convex aspect, just beyond half way, there is a flask-shaped protrusion. The mucous membrane lining this is of the same thickness as in the rest of the appendix. This diverticulum is empty and collapsed—not ballooned out as in *Specimen* 2 (*see Fig.* 69). In fact, there is no ballooning of any of the small pockets in this specimen.

The submucous coat and peritoneal coat are both irregularly thickened. Chief interest lies in the muscularis, which can easily be traced on naked-eye examination.

Near the cæcal end it is well developed, and looks thicker than in the normal appendix (cf. *Fig.* 88). Near the narrowing about the centre of the specimen, there is a considerable local hypertrophy on the convex aspect, with a deficiency opposite. On the mesenteric (concave) border, the muscle coat continues without interruption until the tip is reached. On the convex aspect, however, there is an abrupt end through which the pouch described above passes. On the other side of this pouch the muscle reappears, but becomes practically lost soon after, only a thin irregular line being traceable. In this situation the mucous membrane shows many small pouches, passing out under the serous coat. Near the tip a wedge has been cut for microscopic examination (*Fig.* 71). The mucous membrane shows three very deep crypts, passing deeply into the submucosa, which is very much thickened from fibrosis. The muscle coat showing in the illustration is thick, but very irregular. There is no sharp distinction between it and the submucous coat, and there is a considerable fibrosis around the groups of fibres. The longitudinal coat disappears entirely at one aspect, and a little beyond this, near the crypts of mucous membrane, the circular coat also ceases.

FIG. 71.—*Case* 200. The microscopic appearance of the wall at A, in the specimen illustrated in *Fig.* 70. The muscle coat is partly disorganized by chronic inflammatory changes. A, Submucosa; B, Muscle. (*Mr. S. Steward.*)

SUMMARY.—In this specimen there is chronic widespread inflammation of the wall of the appendix, with a hypertrophied muscle in which gaps are present. Through these gaps the mucous membrane has become everted to form small diverticula. The gaps in the muscle coat are probably the result of fibrosis. There is no distension of either the lumen of the appendix or of the diverticula.

**Specimen 4** (*Case* 201).—The specimen (*Fig.* 72) was removed at operation from a man aged 48 who had suffered attacks of pain for seven years. The specimen has been split along the convex border, and opened out to display the lumen. The appendix is very thick and stumpy. The muscle coat is abnormally well developed, and is interrupted at intervals by septa of fibrous

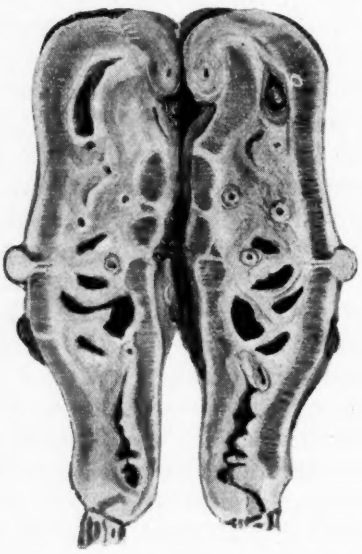

FIG. 72.—*Case* 201. Diverticula of the appendix. The lumen is tortuous and contains fæcal material. (*Mr. A. Edmunds.*)

tissue. Half-way along the antimesenteric margin this fibrous interval has expanded under the peritoneal coat to form a small spherical knob of tissue, which is slightly hollowed out. This hollow communicates by a narrow channel with the interior of the appendix, so that the knob is actually a diverticulum.

The mucous membrane has been thrown into folds so that the lumen, which cannot be traced, crosses and recrosses the mid-line, and has a honeycombed appearance. This appearance is most marked in the middle of the specimen, opposite the small knob-like diverticulum. In this portion the lumen is filled with darkly stained fæcal material.

SUMMARY.—In this case the most marked features are the folds produced in the mucous membrane by shortening of the other coats of the intestine—especially the muscle coat. The latter is thickened, but is interrupted by fibrous septa, at one of which sites a hernial diverticulum has occurred.

**Specimen 5** (*Case* 202).—The specimen (*Fig.* 73) was removed at operation from a man of 27 who had had symptoms of chronic appendicitis for about two years. It consists of one-half of the appendix divided in the long axis in the line of the meso-appendix, and a segment of ileum, to which the tip of the appendix was so firmly adherent that resection was necessary.

FIG. 73.—*Case* 202. Diverticula of the appendix. The lumen is tortuous in the distal half, and sacculated. Adherent to the apex is a segment of the ileum, which was resected at operation. (*Mr. A. Edmunds.*)

Apart from some thickening of all coats, the proximal third of the appendix is not grossly deformed. The lumen, except for the first $\frac{1}{3}$ in., is, however, narrowed almost to the point of obliteration. In the distal two-thirds the mucous membrane is thrown into folds. The spaces between the folds are dilated to a flask shape. These have a narrow communication with the lumen, except at the distal end of the appendix, where the distension is more marked, and the flask-shaped dilatations freely intercommunicate. At the junction of the proximal third and distal two-thirds, the lumen is carried right across the mid-line and passes into a small diverticulum, which has penetrated through the muscle coat. The flask-shaped pouches of the mucous membrane do not penetrate completely through the muscle coat except at one point along the convex border, $\frac{3}{4}$ in. from the tip. The muscularis in contact with the pouches, however, is extremely thin, and it is evident that had the condition been permitted to go on, they would have herniated completely through the muscle coat, owing to pressure atrophy of the latter, and complete diverticula would have resulted.

SUMMARY.—In this case there is chronic inflammation chiefly marked in the proximal third of the appendix, with partial or complete obstruction to the lumen. Distal to this, the mucous membrane is thrown into folds, and the lumen distended so that a series of pouches are formed. The distension is most marked at the tip

of the appendix. At the junction of the proximal third and distal two-thirds, the lumen has been carried towards the concave aspect, and communicates with a small hernial diverticulum.

**Specimen 6** (*Case* 203).—The specimen (*Fig.* 74) was removed at operation from a woman of 30 who had symptoms of acute appendicitis. There had been no symptoms previous to this attack. The specimen consists of one half of the appendix split in the long axis in the line of its mesentery. The proximal portion is acutely inflamed, especially at one point where there is a perforation, surrounded by an area of gangrene. The meso-appendix is thickened from œdema. The lumen of the proximal end is completely obliterated, and in the distal half is distended. Four well-formed hernial diverticula open from it by wide mouths. Two of these are on the meso-appendix aspect, and two on the convex margin. The former two are acutely inflamed. In the intervals between these diverticula, the mucous membrane dips down into the thickened submucous coat, in some cases as far as the muscularis. These deep crypts are probably diverticula in embryo. The muscular coat, though pale from œdema, is very much thicker than usual except at the tip. It is interrupted at intervals by the diverticula, and also by the perforation.

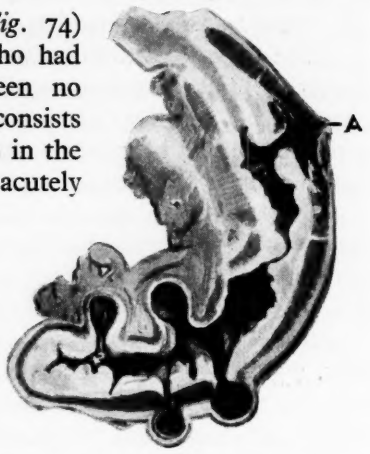


FIG. 74.—*Case* 203. Diverticula of the appendix. The muscular coat between the diverticula is hypertrophied. Perforation (A) has occurred near the cæcal end (? through a diverticulum).

It is impossible to say whether there was originally a diverticulum at the site of perforation. Although there is no history of previous symptoms, the hypertrophy of the muscularis, the thickness of the submucous coat, and the stenosis of the lumen near the proximal aspect all suggest that there had been previous chronic inflammation.

SUMMARY.—An acute appendicitis, in which perforation has occurred, is associated with well-marked hernial diverticula. The proximal part of the lumen is obstructed, and the distal part distended. The muscularis is hypertrophied and there has almost certainly been inflammation of long standing present.

FIG. 75.—*Case* 204. Diverticula of the appendix. Near the cæcal end the whole lumen is carried into a diverticulum. These are all situated on the mesenteric aspect. The lumen contains several concretions. (*Mr. A. Edmunds.*)

**Specimen 7** (*Case* 204).—The hemi-section of an appendix (*Fig.* 75) was prepared from a specimen removed at operation from a woman of 28 who had typical symptoms of acute appendicitis. The proximal half of the appendix is acutely inflamed, and the meso-appendix is thickened from œdema. The proximal part of the lumen is narrow but not obliterated, and just beyond this point the *whole lumen* is carried from the mid-line through the muscle coat to form a diverticulum.

Several diverticula, varying from a wedge-shaped crypt of the mucosa to a fully formed flask-shaped pouch, pass through the muscle coat into the meso-appendix. These are *all* on the concave aspect of the appendix. The muscle coat is somewhat hypertrophied, especially between the diverticula. There is no interruption of its fibres on the side away from the meso-appendix. Six concretions lie in the lumen of the appendix, but the diverticula are empty. The submucous coat is not thickened and there is no evidence of previous gross inflammatory changes.

SUMMARY.—An acutely inflamed appendix is associated with the presence of many diverticula, herniated through the muscular coat on the side of the meso-appendix. The *whole lumen* is carried into the first diverticulum, thus producing obstruction distal to it, so that concretions are present in the lumen. There has probably not been any gross inflammation of the appendix previous to the acute attack.

**Specimen 8** (*Case* 205).—This specimen (*Figs.* 76, 77) was removed at operation from a man of 21 who for two years had suffered from symptoms of chronic appendicitis. A giant congenital diverticulum was removed from this patient. Unhappily he developed ileus after the second operation, which was difficult owing to the position and fixity of the appendix, and died.

*Fig.* 76 shows one-half of the appendix divided longitudinally, and *Fig.* 77 the distal portion of the other half, cut across the long axis. The remaining material was utilized for microscopic investigation.

FIG. 76.—*Case* 205. Diverticula of the appendix. The wall is thickened, and the lumen distended into saccules. (*Mr. G. B. Davies.*)

FIG. 77.—*Case* 205. A section through the specimen shown in *Fig.* 76 near the distal end, and passing through a large diverticulum. (*Actual size.*)

The whole appendix is greatly thickened and somewhat kinked. The meso-appendix is loaded with fat and bunched up into the angle formed by the kink. The lumen of the proximal half is normal. Opposite the bend in the appendix it disappears from view. This is probably because it has migrated to one or other side of the mid-line, as in some of the previous specimens. As the angle is passed, the lumen reappears as a small pocket to the left of the mid-line, but within the muscle coat. Distal to this, it forms three large pockets, separated by thick ridges of fibrous tissue. Even after serial sections of the opposite half of the appendix in the region of the pockets, it is difficult to trace the lumen between these dilatations. On the half cut away there is a large diverticulum, shown in transverse section in *Fig.* 77.

Except for an area on the concave border opposite the kink, the muscular coat is thickened and prominent. In the former situation it appears to be infiltrated with fat and fibrous tissue, and cannot be made out.

The diverticulum is situated on the concave border, just anterior to the meso-appendix. *Fig.* 78 is a low-power drawing of a section through the diverticulum and the adjacent wall of the appendix.

The diverticulum has no trace of muscle-fibres in its wall, with the exception of a muscularis mucosæ to be seen on high magnification. The submucous coat consists of loosely woven fibrous tissue, in which a large blood-vessel runs a

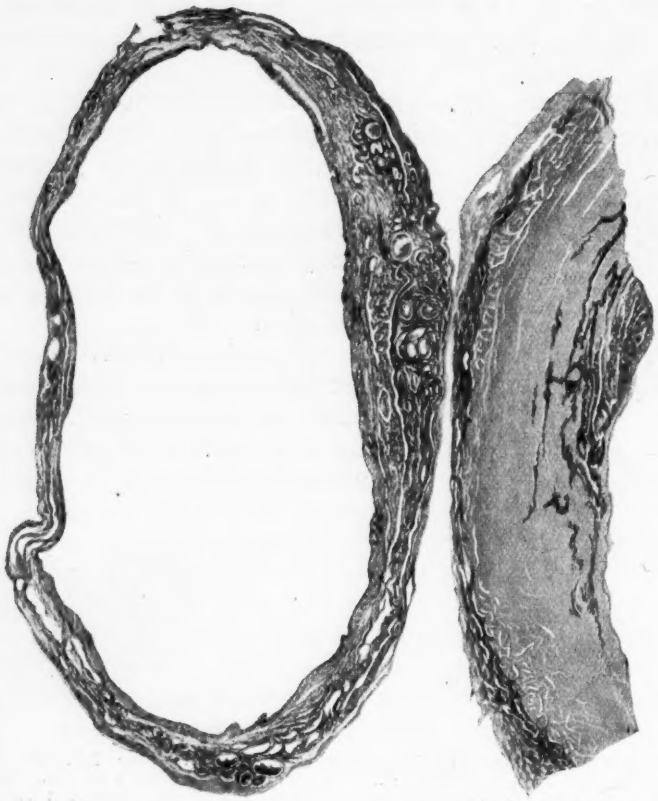

FIG. 78.—*Case* 205. The microscopic appearance of the diverticulum illustrated in *Fig.* 77. Only a few muscle fibres are to be seen in the wall of the diverticulum. (Van Gieson stain.) (× 5.) (*Mr. S. Steward.*)

tortuous course. This vessel is best seen on the wall next to the appendix, where the submucosa is at its thickest. The communication between the diverticulum and the lumen of the appendix cannot be seen in this field or in any of the sections taken. It is a very narrow passage, a faint indication of which is seen in the macroscopic view.

The striking feature of the appendix wall adjacent to the diverticulum is the extreme thickness of the muscle coat. It has taken the stain (van Gieson) very poorly, however, and is a much lighter colour than normal. In the higher-power examination there is a lack of definition between the fibres and a scarcity

of nuclei, and there appears to be an inflammatory exudate between the groups of fibres.

SUMMARY.—The specimen shows an appendix thickened as a result of chronic inflammation. The distal half of the lumen is tortuous, and distended to form a series of pockets, which are retained within the muscle coat. Near the tip is a large diverticulum, whose opening corresponds in situation with a large blood-vessel. The muscularis throughout the appendix, except at one situation where the appendix is kinked, is thickened, probably from hypertrophy, and has undergone a secondary degenerative process. The diverticulum possesses no muscular coat.

FIG. 79.—*Case* 206. The microscopic appearance of *Specimen* 9. The muscular coat of the appendix is extensively broken up by inflammatory fibrous tissue. A, Fibrous tissue; B, Muscle coat.

**Specimen 9** (*Case* 206).—This specimen (unillustrated) was removed at operation from a woman of 48. It was a short, very stumpy appendix with the walls extremely thickened by fibrous tissue. There were five or six small swellings under the peritoneum in the distal half, with rigid walls. On section the lumen was almost completely obliterated by fibrosis, and the small nodules were solid masses of fibrous tissue. The muscle coat was broken up extensively by fibrous masses extending from the submucosa.

The microscopic appearance in this case is illustrated by a photomicrograph in *Fig.* 79. There is an extensive fibrosis of the submucosa, which has penetrated into the muscle coat, subdividing into small atrophic islands. In the centre the whole thickness of muscle has been invaded by fibrous tissue, and all that remains of this coat is small isolated bundles of fibres buried in great masses of fibrous tissue. There is no clear division between submucosa and muscularis.

SUMMARY.—The case illustrates the way in which an inflammatory fibrosis may cause great gaps in the muscle coat through which diverticula may occur.

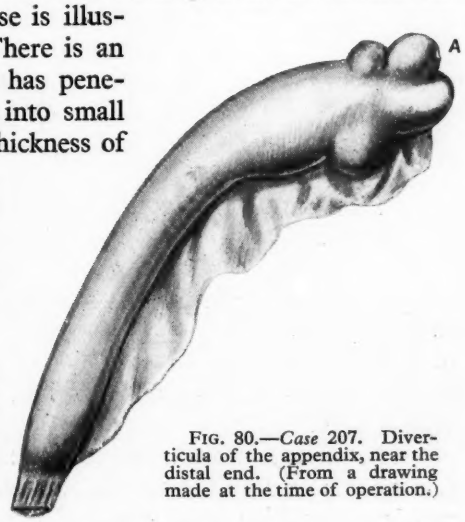


FIG. 80.—*Case* 207. Diverticula of the appendix, near the distal end. (From a drawing made at the time of operation.)

**Specimen 10** (*Case* 207).—The specimen illustrated in *Fig.* 80 was removed at operation from a man of 55. The appendix is thickened and almost bulbous

towards the distal end. Near the tip are three small diverticula, one of which passes into the meso-appendix, and the other two lie on the free border.

*Fig.* 81 illustrates a microscopic section passing through the tip of the appendix and the diverticulum marked A in *Fig.* 80. The lumen of the mucous

FIG. 81.—*Case* 207. Microscopic appearance of a whole section across the tip of the appendix illustrated in *Fig.* 80. The diverticulum opens from the antimesenteric border of the appendix, and contains very little muscle tissue in its wall. The lumen of the appendix is eccentric. (*Mr. S. Steward.*)

membrane is eccentric, and for half its diameter lies in a diverticulum of the normal type.

The muscular wall of the appendix is well developed. The diverticulum passes through a gap in the longitudinal muscle coat, carrying with it the circular muscle coat, which is extremely thin toward the fundus of the diverticulum. The submucous layer is thicker here than at the neck.

*Fig.* 82 illustrates a section from another aspect. It has caught the lower border of the diverticulum. Here the lumen of the appendix is no longer eccentric. The most remarkable feature of this section is the integrity of the circular muscle-fibres in contact with the diverticulum, and the way the longitudinal fibres of the appendix are carried out into the diverticular wall. Note the unusually large gap in both coats at the attachment of the mesentery.

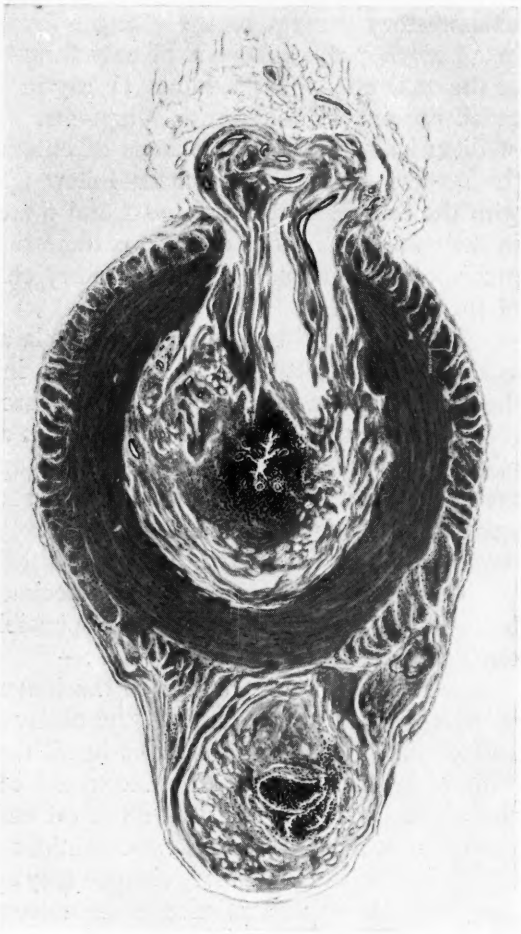

FIG. 82.—*Case* 207. A further section from *Specimen* 10, *Fig.* 80. The arrangement of the muscular fibres at the base of the diverticulum is well shown. The vascular gap on the mesenteric aspect of the appendix is unusually wide. (*Mr. S. Steward.*)

SUMMARY.—The mucous membrane has been bodily displaced to one side, and has herniated through a gap in a well developed muscular coat.

## PATHOGENESIS

Before arriving at conclusions from the abundance of data offered by these ten specimens, it will be well to attempt a grouping, and to discuss the significance of each group. These are discussed in the following order: (1) The relation of the diverticula to the muscular coat; (2) The inflammatory changes in the appendix wall; (3) The situations of the diverticula in relation to the circumference of the appendix; (4) The changes in the mucous membrane affecting the lumen.

**1. The Relation of the Diverticula to the Muscular Coat.**—In all cases the fully formed diverticulum lacks a muscular coat. In the greater number of cases the diverticulum is a true hernia through a gap in the muscle coat. In other cases, such as in *Specimen* 5, where there is distension of the lumen so that pockets are formed, there still remains a thin layer of muscle, and there can be no doubt that these pockets are the direct result of distension. They cannot be regarded as fully developed diverticula, but rather as sacculations of the lumen. Eventually, if the process were allowed to go on, the muscular coat would completely disappear over the fundus as a result of atrophy, and a fully developed diverticulum, to be seen as a prominence under the peritoneal coat, would result. Thus there are clearly two types of diverticula: (*a*) Hernial pouches of mucous membrane forced through a gap in the muscle coat; and (*b*) Distended pockets of the mucous membrane over which the muscularis will eventually atrophy, so that a complete diverticulum, visible from the peritoneal aspect, is formed.

**2. Inflammatory Changes in the Appendix Wall.**—All show inflammatory changes. In 7 there is chronic inflammation and in 2 of these acute inflammation has been superadded. In *Specimen* 1 the inflammation appears to have been acute from the start, but one cannot be certain of this point. The duration of the history must be given second place in importance in deciding the chronicity of the inflammatory process to the changes found on pathological examination. Thus in *Specimen* 7 the history is of only forty-eight hours' duration, but the presence of the concretions in the lumen clearly indicate that pathological change had preceded the onset of subjective symptoms. There is no evidence that the presence of diverticula has been the cause of chronic inflammation. Everything points to the reverse—that the diverticula follow upon inflammation. All of the pouches with the exception of *Specimens* 1 and 6 are free from gross inflammatory changes in their walls. In none of them is there any retention of fæcal material that might predispose to the onset of inflammatory changes such as occur with diverticulosis of the colon.

In *Specimen* 6 two of the diverticula are acutely inflamed, but this does not establish their guilt as the cause of the initial inflammation. It is possible that the perforation in this case took place through a diverticulum, although the area is so destroyed that one cannot be certain on this point. That acute diverticulitis might supervene in diverticulosis of the appendix is, of course, conceivable, and even a likely possibility. Wilkie reports such a case. But this fact is no evidence against the thesis that chronic inflammatory changes in the appendix precede the development of diverticula.

Chronic inflammation in these specimens, at all events in *Specimens* 2 to 10, has aided, possibly been mainly responsible for, the formation of diverticula in one or more of the following ways :—

*a*. By causing obstruction to the lumen of the appendix. This has occurred in *Specimens* 2, 3, 5, 6, and 8, the obstruction being somewhere in the proximal half of the lumen. The stenosis in all these cases is marked, but not complete. This is in accordance with what would be expected, as complete obstruction to the appendix at a point near its cæcal end would be more likely to result in a mucocele rather than cause the conditions present in these specimens.

*b*. Chronic inflammatory changes may act by causing weak areas in the muscular coat, through which herniation of the mucous membrane may occur. The sequence of events is probably this. The abundant lymphoid tissue normally present in the appendix is infected, and granulation tissue appears, which invades the submucous coat, and finally the muscularis, leaving a weakened scar. A significant fact is that in many of the microscopic sections made from these specimens the lymphoid tissue is either very scant or absent entirely.

In *Specimen* 3 the irregular sites of the small diverticula suggest that the weak areas in the musculature were provided by changes due to chronic inflammation, for they show no tendency to follow the gaps provided by the entrance of the blood-vessels, which will be described subsequently. The microscopic section of *Specimen* 9 illustrates how the muscular coat may become disorganized by chronic inflammation.

*c*. Chronic inflammation may be the cause of persistent spasm in the muscular coat, the significance of which is discussed subsequently.

**3. The Situations of the Diverticula in relation to the Circumference of the Appendix Wall.**—The most favoured site for the development of diverticula

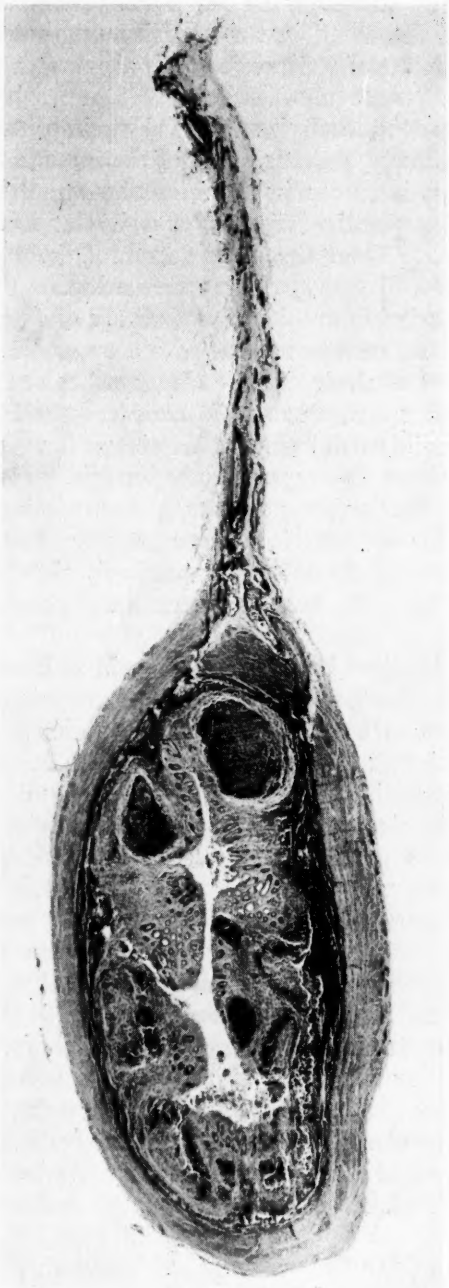

FIG. 83.—A photomicrograph of a section through the appendix and its mesentery to show the entry of the vessels through the muscular coat. (*Mr. S. Steward.*)

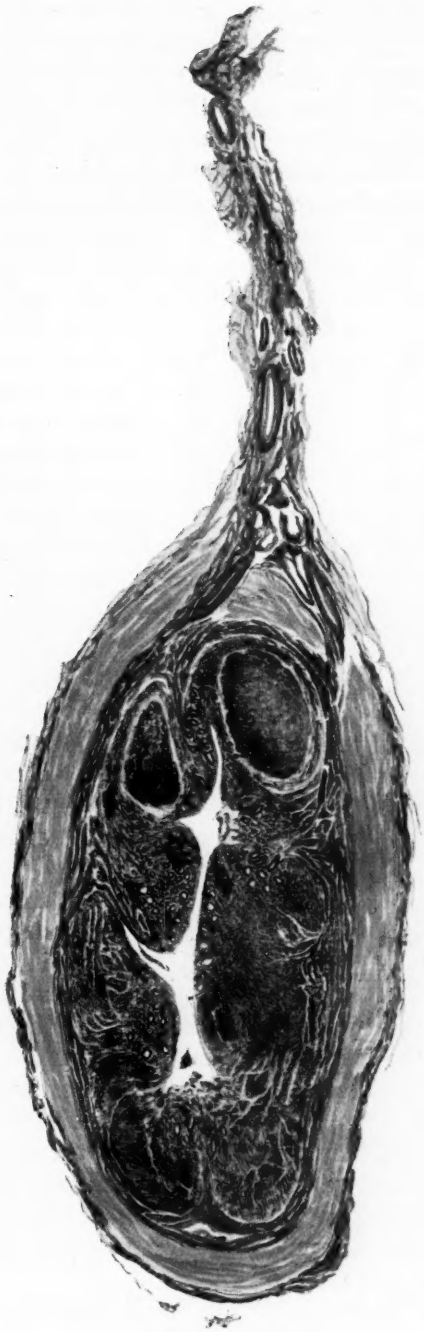

FIG. 84.—Drawing to show more clearly the anatomy of the section illustrated in *Fig.* 83. (*Miss Barclay-Smith.*)

is along the concavity of the appendix, along which the vessels of the meso-appendix enter. This is most clearly illustrated in *Specimens* 1 and 7. The next most frequent site is on the opposite (convex) margin.

The gaps caused in the musculature by the entry of the blood-vessels are similar in anatomical features to those of the small intestine. The number of final branches arising from the appendicular arteries depends upon the length of the appendix, varying between 4 and 12.

Immediately before reaching the appendix, each branch divides into two, which pierce the muscle coat, somewhat obliquely, on either side of the mesenteric line. This is shown in the microphotograph of a normal appendix (*Fig.* 83) and in the illustration made from the same section (*Fig.* 84). *Fig.* 85 is a diagrammatic representation.

FIG. 85. — Diagrammatic representation of the mode of entry of the blood-vessels through the muscle coat.

The relation to the entry of vessels of diverticula at the convex margin of the appendix is not so well marked. Before the branches of the appendicular artery enter the concave aspect of the appendix, small branches are given off, which pass on either side around the appendix underneath the serous coat, and enter the muscle layer on the convex margin. According to Stout, these branches do not pass completely through the muscle layer, but terminate in supplying the fibres.

**4. Changes in the Mucous Membrane Affecting the Lumen.**—The most remarkable observation to be made from these specimens is the behaviour, in some of the cases, of the lumen of the appendix.

In *Specimens* 4 to 8 the mucosa has been thrown into folds. In between these folds are deep crypts, which in some cases—for example, in *Specimen* 4—have been distended to form wide-mouthed pouches. What is the explanation of this pleating of the mucosa? There clearly has been a relative increase in length of the mucosa and submucosa coat as compared with the length of the muscular and serous coats. Is this the result of actual lengthening of the mucosa or of shortening of the subjacent muscularis? There is no reason to think that the mucosa should increase in length, and the appearances support the view that the folds in the mucosa are due to shortening of the muscular coat. Under normal conditions the attachment between the submucous coat and the muscularis is by loose bands of areolar and connective tissue, so that there can be a considerable excursion of one upon the other.

The muscle coats in all these specimens (4 to 8) is very much thicker than in the normal. In ten normal appendices the average thickness of the muscle coat determined by measurement with fine-pointed dividers was approximately 0·75 mm. In these five specimens, the muscle layer is much thicker, varying between 1·5 and 2·5 mm. This increase in thickness may be due to at least one of three

causes: (*a*) Contraction of the muscle and fixation in contraction; (*b*) Hypertrophy; and (*c*) Inflammatory œdema.

The last-named certainly occurs, but cannot be responsible in all the cases, and one is left with (*a*) and (*b*). If this thickness is due to hypertrophy, one would expect to find a partial obstruction present in every case, and the alternative explanation must therefore be accepted. The pouching of the mucous membrane, which may lead to diverticula, is due to shortening of the muscle coat as a result of persistent contraction. To thickening of the coat from this cause must be added a secondary hypertrophy in most of the cases, in which obstruction has been caused by the formation of these folds in the mucous membrane. Migration of the whole lumen to one or other side is associated with this pouching in *Specimens* 4, 5, 7, and 8. It is best shown in 5 and 7. Here both layers of the mucous membrane are carried right through the muscle coat into a diverticulum. In both cases this has caused a partial obstruction, with subsequent distension of the lumen distal to this point. This phenomenon of migration of the lumen is difficult to explain. It clearly cannot result from high pressure inside the lumen, and no outside traction could drag both surfaces through the muscular wall of the appendix. One must therefore turn to aberrations in the normal action of the muscularis as the cause. The probable way in which this acts is demonstrated by diagrams in *Fig.* 86.

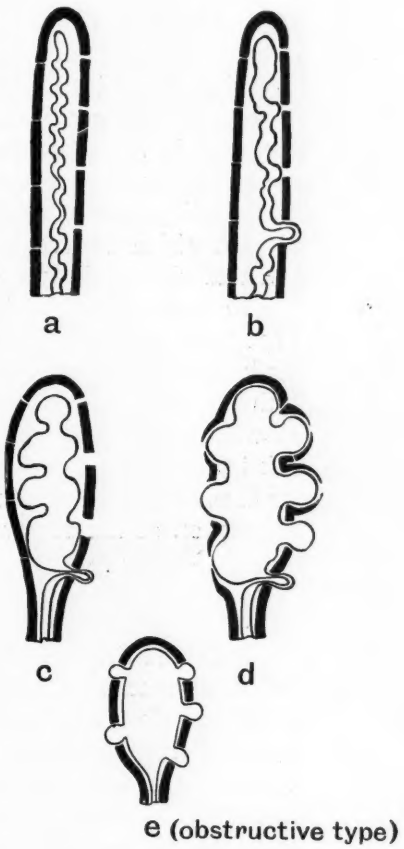


FIG. 86.—The mechanism of formation of diverticula of the appendix.

There is first tonic spasm of the longitudinal fibres, so that the mucous membrane is thrown into folds. Such spasm of the longitudinal fibres will increase the vascular gaps. Spasm of the circular muscle now follows, and the potential cavity of the appendix is obliterated, the surfaces of the lining mucous membrane being pressed together, and lying in contact. When such spasm occurs, the loose folds of mucous membrane, lying in contact, will seek an outlet, and both surfaces may pass into a gap occasioned by the passage of a blood-vessel on the concave aspect. Once this has been achieved, there will be a tendency for it to become permanent, and by causing a marked obstruction to the outflow of contents from the lumen distal to it, to cause distension of the lumen with pouching of the folds of mucous membrane. Such distension may cause herniations through vascular gaps, or cause thinning of the muscularis over the pouches from atrophy so that fully developed diverticula may be formed from them.

*Specimen* 7 illustrates perfectly how crypts of mucous membrane caused by contraction of the longitudinal muscle may pass through the gaps in the musculature on the concave aspect.

Such spastic conditions of the musculature may result from lack of neuromuscular co-ordination, such as seems to form diverticula in the small and

large bowels, or may have a more local origin in chronic inflammation of the appendix wall.

**Commentary.**—From this seeming tangle of facts, the explanation of the pathogenesis of diverticula of the appendix begins to unfold itself. In not all the cases are the mechanics of production the same, and we can distinguish at least two types:—

1. A hernial protrusion of the mucous membrane through a gap in the musculature, occasioned in most cases by the passage of a blood-vessel, and in some as a result of local degeneration of the muscle.

2. Distension of sacculations in the mucosa, with subsequent thinning from atrophy of the muscularis covering them.

The first type may arise purely as a result of partial obstruction to the lumen. *Specimen* 2 is an excellent example of hernial diverticulum as the result of obstruction. The second type—sacculation of the mucous membrane—is a more intricate process, and is indirectly due to spasm of the muscularis, with tortuosity and migration of the lumen (*Specimens* 4 to 8, and ? 3). Subsequently, in these cases, obstructive effects are produced, so that the mucous membrane may be forced into gaps in the musculature. Thus diverticula of the appendix may be due to one of two exciting causes: (1) Passive distension; (2) Irregular muscular action. The predisposing causes are: (1) The presence of gaps in the muscular coat through which the vessels enter; and (2) Weakening of the muscular coat as the result of chronic inflammation.

In both the cases diagnosed radiologically the whole colon is also affected. That there is no obstruction to the appendix acting as a cause of pressure within the lumen is proved by the entry of barium. The formation of the diverticula in both the colon and the appendix is undoubtedly due to the same factors, and the knowledge gained of the myogenic origin of diverticula of the appendix is a further link in the chain of evidence that the author has brought forward to substantiate the thesis that hernial diverticula of the alimentary tract owe their origin to irregular contraction of the muscularis (*see* BIBLIOGRAPHY, p. 107).

## THE LITERATURE

Pack and Scharnagel describe a case with thirty-six diverticula, and concluded that these resulted from an exudative inflammation which caused disorganization of the muscle coat. Wilkie describes two cases associated with obstructive carcinoma of the appendix. In one of these perforation occurred through a diverticulum. Chase thinks the gap in the musculature—which he calls "hiatus muscularis"—may occur from the passage of smaller vessels and lymphatics or be an interval occupied solely by areolar tissue or fat. He recognizes two types: simple herniation of the mucous membrane through a gap, and diverticula due to chronic inflammation. The article of MacCarty and McGrath has already been referred to. Stout has shown by experiment upon the living dog that if a gap be made in the muscular wall of the appendix, the mucosa and submucosa are protruded. In one such experiment he excised a portion of the serous coat and muscular coat measuring 6 mm. by 8 mm. The appendix contracted both in diameter and length and became very hard, and the mucous membrane was forced through the gap to form a prominence 4 mm. in height. When the muscle coat

relaxed again the mucous membrane pouch became smaller, but did not disappear completely. Moderate hydrostatic distension made the diverticulum stand out more clearly as a smooth rounded hemisphere. When the hydrostatic pressure was increased further, the coats of the appendix slowly thinned from stretching, and the gap in the muscularis became wider. The diverticulum grew steadily less until it disappeared entirely. Such an effect is produced by the internal pressure overcoming the force of muscular contraction.

From this and similar experiments Stout concluded that diverticula may be produced both by muscular action and by increased intraluminal pressure. These views correspond with the deductions made from the author's series of pathological specimens.

## DIAGNOSIS AND TREATMENT

There are no symptoms directly referable to the presence of diverticula, so that it is not possible to diagnose the condition prior to operation. Only

FIG. 87.—*Case* 209. Diverticula of the appendix, demonstrated radiologically. Diverticula were also present in the colon. (*Dr. Graham-Hodgson.*)

very rarely, as in the two cases described, do diverticula show up on X-ray examination. The appearance of one of the cases is illustrated by a skiagram

of a barium meal taken thirty-six hours after ingestion (*Fig.* 87). The treatment is appendicectomy.

## UNUSUAL FORMS AND PSEUDO-DIVERTICULA

*Fig.* 88 illustrates an appendix in which there is a narrowing of the lumen ⅓ in. from the tip. The appendix is otherwise normal. The distal pocket is somewhat distended and its wall a little thickened. The case cannot be regarded as a diverticulum in the true sense of the word. There is no 'turning aside' (*Case* 208).

The illustration in *Fig.* 89 was copied directly from an article by Herb. The excuse offered for making such a copy is that words could not readily convey the

FIG. 88.—A pseudo-diverticulum of the appendix. The distal part of the lumen is shut off by a transverse band. (*Miss Barclay-Smith.*)

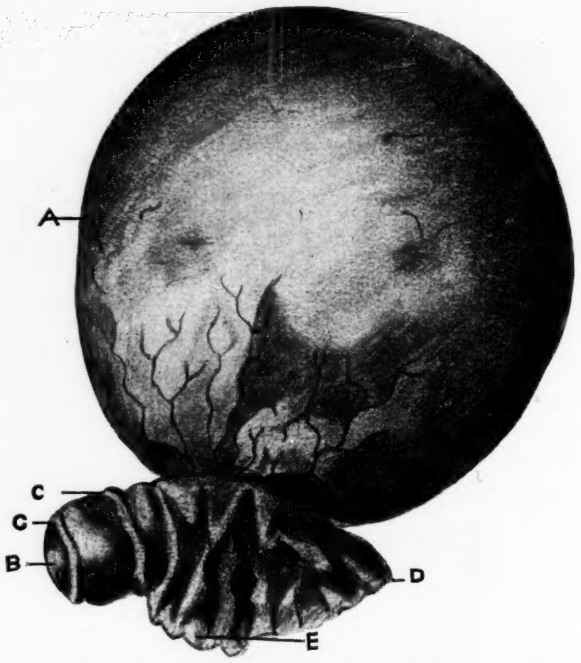


FIG. 89.—An enormous diverticulum of the appendix, copied from an illustration of the case described by Herb. A, Diverticulum; B, Proximal end of appendix, showing closure; C, Retracted muscular coats; D, Distal end of appendix; E, Meso-appendix. The lighter areas on the diverticulum are calcified plaques.

remarkable condition which is pictured. The enormous swelling arising from the convex surface of the appendix is 9 in. in circumference, and communicates with the lumen of the latter by a circular opening 1 in. wide. The lumen of the appendix is obstructed at the proximal end. The sac was filled with jelly-like substance. Its wall consists of mucosa and fibrous tissue only, there being no muscle and no mucous membrane. The muscularis of the appendix ends abruptly at the margin of the sac. This, though unquestionably a type of diverticulum, belongs to the little understood condition described as mucocele of the appendix.

My thanks are due to the Council of the Royal College of Surgeons for permission to publish this section of the Jacksonian Prize Essay for 1932.

I wish also to thank my colleagues on the staff of King's College Hospital for permission to use cases operated upon by them, and Miss Mary Barclay-Smith and Mr. S. Steward, who are responsible for the majority of the illustrations.

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# ACUTE ENTERIC INTUSSUSCEPTION IN AN ADULT CAUSED BY A LIPOMA

## WITH A SURVEY OF THE LITERATURE

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THE case described below warrants publication in that it presents the following features: (1) It is a case of intussusception in an adult; (2) It is of the pure enteric variety, small bowel only being involved; (3) The cause is a benign tumour, a submucous lipoma; (4) The specimen (*see Fig.* 90) illustrates the modern view of the physics of intussusception; (5) After resection and entero-anastomosis the patient made a complete recovery.

## CASE REPORT

T. B., aged 52 years, a coal heaver, was admitted to Boundary Park Municipal Hospital, Oldham, on Sunday, March 8, 1931, suffering from intestinal obstruction.

HISTORY.—The patient's previous medical history was unimportant except for an operation for anal fissure in 1928. He gave a history of having been taken ill on the previous Thursday with severe cramp-like pains in the abdomen, and he went to bed on that day. He vomited frequently and the pains became worse. He had passed neither fæces nor flatus since the onset of his illness.

ON EXAMINATION.—He looked ill and 'toxic', and at midnight his temperature was 97°, the pulse 92, and the respiratory rate 20. The abdomen was distended, tender all over, and liver dullness had disappeared. There was dullness in both flanks. Nothing could be felt per rectum and the examining finger was not blood-stained. Immediate operation was decided upon.

OPERATION.—Under a general anæsthetic a mass could be felt to the right of and below the umbilicus. The abdomen was opened through a right paramedian incision and a large quantity of blood-stained fluid escaped. The mass proved to be an intussusception composed entirely of small intestine. The intussuscipiens was gangrenous and covered with plastic lymph. A very gentle attempt at reduction caused the outer coat to tear badly. Resection was decided upon, and the whole mass was rapidly removed and a lateral anastomosis made between two convenient loops of small intestine. The abdomen was then swabbed out. As the patient showed signs of collapse no attempt was made to close the abdomen in layers, six silkworm gut through-and-through sutures only being used. The usual measures were employed in the immediate after-treatment.

SUBSEQUENT PROGRESS.—The patient was very ill for some days, severe incontinence being the most striking feature. Anti-gas-gangrene serum was given daily for a week. The incontinence gradually subsided and the wound broke down and discharged in several places, but at the end of a week the patient began to improve, the wound commenced to heal, and the bowels became normal in action.

He was discharged from the hospital soundly healed, feeling well and able to walk, six weeks after admission. He is now engaged in his former occupation as a coal heaver and feels perfectly well.

PATHOLOGICAL REPORT (*Fig.* 90).—The tumour projecting into the lumen of the small intestine has a nodular surface. It is attached by a pedicle of fibrous tissue and is enclosed

in a fibrous capsule. On section it is pale yellow in colour and firm in consistence. It is roughly spherical in form and about 2 cm. in diameter. The mesentery is inflamed and there are definite signs of peritonitis. Histologically the tumour is a pure lipoma, staining uniformly with Sudan III.

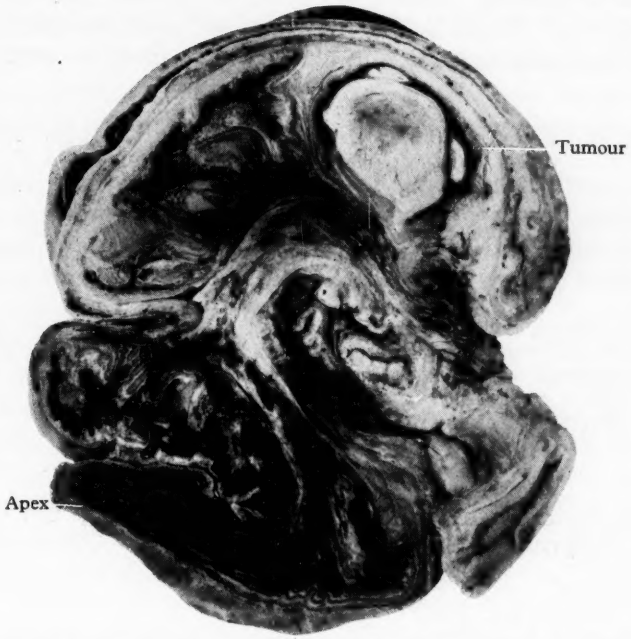


FIG. 90.—A portion of the intussuscipiens is not distinctly shown in the photograph. As indicated in the report of the case, the receiving layer was gangrenous and tore on an attempt at reduction, and whilst in the preserving fluid the torn and gangrenous portion curled away behind the plane of section.

## INTUSSUSCEPTION IN ADULTS

To those associated with large teaching and children's hospitals intussusception is fairly common, yet taking the ordinary incidence of the condition in relation to the population it is rare. The writer is acquainted with a very able general practitioner who never even saw a suspicious case in twenty-five years' practice.

During the last decade there have been 27 cases (one of which recurred) operated upon at the Oldham Royal Infirmary, 10 cases at the Boundary Park Municipal Hospital, and 1 at a local nursing home—a total of 38 cases in ten years amongst a population of about a quarter of a million served by these institutions. Of these cases, only one, the writer's, was an adult.

Choyce[4] states that not more than 12 per cent of all cases of intussusception occur in patients over 10 years old. Glover,[11] in reporting a case of enteric intussusception in a man of 47 with no indication as to cause, comments on the rarity.

The majority of cases of invagination in adults appear to be due to a growth of a benign character. Muller[26] describes one as due to a mucous papilloma. Davies[6] and Dewis[8] describe cases due to fibroma, and Somerville-Large[33] one due to a polypus. As a malignant cause Morrison[25] has recorded a case due to a lymphosarcoma. Lenner[18] and McIver[20] have each described cases due to Meckel's diverticula. Ruppaner[32] quotes a very unusual one following gastro-enterostomy.

Mesnager[22] notes a case apparently due to a fall, and refers to a similar case recorded by Kopp. Mooro[24] describes two cases occurring in Mohammedans during a fast, and points out that excessive hunger associated with the anticipation of a meal can excite abnormal peristalsis. A case due to a tuberculous intestine in a boy of 16 has been recorded by Sussig,[37] whilst Thorleifson[38] has described a case of intussusception of the jejunum where no cause could be found at autopsy.

## LIPOMATA OF THE GASTRO-INTESTINAL TRACT

There is much overlapping in the literature connected with this subject. Previously reported cases have been overlooked by some writers, so an attempt is made below to collect and correlate reports from all sources. Using as a basis the paper by Comfort,[5] the writer was able to trace the following :—

| | |
|---|---|
| Comfort, own and collected cases .. .. .. | 96 |
| Mirolli,[23] cases not included above .. .. .. | 103 |
| Stettin,[35] cases not included above .. .. | 14 |
| White and Judd,[40] cases not included above .. | 13 |
| Writer's case and other cases not included above .. | 16 |
| Total .. .. | 242 |

The cases found by the writer are reported by Bailey,[1] the BRITISH JOURNAL OF SURGERY SUPPLEMENT,[2] Brandes,[3] Forni,[10] Farr,[9] Horsley,[12] McCloskey,[19] Matry,[21] Neumann,[27] Oughterson and Cheever,[28] Polya[30] (two cases), Rouse and Mekie,[31] and Stewart and Illick[36] (two cases).

The anatomical distribution of the tumours was as follows :—

| | | | |
|---|---|---|---|
| Stomach .. .. | 27 | Sigmoid colon .. | 21 |
| Duodenum .. .. | 18 | Rectum .. .. | 11 |
| Small intestine .. | 64 | Mesentery .. .. | 1 |
| Ileocæcal region .. | 10 | Incomplete accounts .. | 11 |
| Cæcum .. .. | 20 | | |
| Colon .. .. | 59 | Total .. .. | 242 |

Intussusception is reported to have occurred in 80 cases in the following sites :—

| | | | |
|---|---|---|---|
| Small intestine .. | 34 | Sigmoid colon .. | 10 |
| Ileocæcal region .. | 5 | Uncertain .. .. | 5 |
| Cæcum .. .. | 6 | | |
| Colon .. .. | 20 | Total .. .. | 80 |

It will be seen therefore that not only is lipoma of the gastro-intestinal tract an uncommon condition, but also that enteric intussusception due to this benign new growth is very rare.

## MECHANICS OF INTUSSUSCEPTION

Our surgical forefathers cannot be blamed for the unwieldy name given to this condition. A 'reception within' warrants some preparation and something to receive. The writer has been unable to trace any case where a foreign body has caused invagination; a foreign body is either passed per rectum or it causes perforation or obstruction of the bowel. It would appear, then, that it is only

when that which is to be received is part of the bowel itself or attached to it that actual invagination occurs, and this presumption is borne out not only by the physiological but also by the pathological facts.

Starling[34] describes normal peristalsis as a slowly moving wave, generally at about 2 cm. a minute, with contraction of the gut above the food mass and relaxation below it. Illingworth and Dick[16] believe that there is considerable evidence to show that the underlying cause of intussusception is some derangement of this peristaltic mechanism excited in children by errors in diet. Such errors are notorious between the fourth and seventh month, but it is also pointed out that the inhibitory nervous apparatus is developed last and that its action is apt to lag behind that of the motor apparatus. In infancy, therefore, the inhibiting activities of the parasympathetic nerves are functionally weak and the tonicity of the plain muscle outweighs, for a time at least, its capacity for relaxation.

Comparing the intussusception of the infant with that of the adult, Hubner[13] points out that intestinal disorders in the infant, such as colitis, seem to have no great influence, as nearly all cases occur in infants in good health. A *terrain maladif* is not necessary as a background. He quotes Withner, who noted that a tumour was found in only 2 cases out of 200 infants suffering from intussusception. Hubner believes that if a peristaltic wave starts at a given point it is accompanied always by an inhibition in the segment below this point. This lower segment relaxes under the influence of a stimulus brought upon the given point, whilst above the point there is a reinforcement of the contraction. With a normal intestinal content only a minimum muscular effort is required, contraction following upon dilatation, and so on. If, however, from some cause the ring of contraction becomes fixed and immobile, one can conceive that this fixed and immobile part of the intestine can penetrate into the dilated portion below it and so form the beginning of the invagination. Nothnagel is quoted as having actually produced invagination by electric stimulation of the bowel itself. Therefore "the *agent provocateur* of the exaggerated peristalsis on the one hand is, on the other, the fixing agent."

Dewis[8] noted that while the tumour is usually situated near the apex of the invaginated mass, it is occasionally attached to other portions of the mass. It is pointed out by Wardill[39] that when a polyp of bowel is associated with an intussusception there is a definite relationship of cause and effect. He has found that specimens show that tumours may occupy a position some considerable distance proximal to the apex although still included in the intussusception. (This important point is very well illustrated in the writer's own case.) Mere traction on the polypus by intestinal movements does not account for the commencement of the invagination. The tumour lies within the intestinal lumen and so acts as a foreign body which produces spasmodic contraction of the gut around it with inhibition of the gut immediately distal to it. "The conditions are now favourable for that act of peristaltic gymnastics whereby the contracted part is induced to slip into the dilated portion." Here is the whole physiological and pathological explanation in a nutshell.

Discussing the problem at a later date, Iason and Filberbaum[14] go into the causes of intussusception in adults. Accepting Wardill's theory, they state that other causes may be: (1) Passive, due to the mere weight and pull of the tumour; (2) Active, owing to the violent peristalsis due to the normal reaction to a foreign

body in the lumen; (3) Perverted muscle action due to the presence of a tumour; and (4) Paralysis of the bowel itself.

Ibos and Legrand-Desmons,[15] in discussing a case of retrograde intussusception of the small intestine, are of the opinion that their case proves that a pathological antiperistalsis can occur in the intestine.

## DIAGNOSIS

The diagnosis of a benign new growth in the alimentary canal must always be a matter of difficulty, and usually its presence is not suspected until a laparotomy is performed for some condition of which it may or may not be the cause. Pollosson and Rougemont[29] have described a syndrome which is worth quoting as it offers a fairly definite clinical picture. The patient has several attacks of abdominal pain of such severity that he remembers each attack. He suffers from borborygmi and the colicky pains come on at regular intervals. There is complete abdominal relaxation with no distension and no rigidity. The pulse and temperature remain normal. If a tumour is felt at this stage, appendicitis or a similar inflammatory condition can be ruled out, as such conditions cannot form a swelling in a few hours. Examination of the urine may help to exclude renal colic. These writers emphasize the importance of abdominal palpation under an anæsthetic. (This proved of value in the writer's own case.) With the onset of hæmorrhage, obstruction, or intussusception the symptoms due to these conditions will appear.

In this connection Wardill[39] stresses the point that when operating for intussusception, particularly in subjects over the age of 2 years, a careful examination should be made of the *proximal healthy bowel* (the italics are mine) for possible new growth. Hubner[13] makes a similar observation, but states that in all cases where an intestinal tumour was noted, an acute intussusception was the first sign of its existence. Iason and Filberbaum[14] point out that the appearance of blood per rectum is less frequent in the intussusception of adults than in that of children, and if present points to torsion or ulceration of the tumour. (It will be noted that the examining finger showed no blood in the writer's case.) The probable explanation of this is that when the invagination is high up coagulation takes place.

Before passing from the consideration of the diagnosis of benign intestinal tumours it is well to remember that these tumours are often discovered during the so-called 'cancer age'.

## TREATMENT

In the treatment of intussusception Leclerc[17] recommends attempts at disinvagination in the first place, and, if this fails, resection either of the invaginated cylinder of gut or of the whole of the affected bowel with entero-anastomosis. In desperate cases the formation of an artificial anus or 'exteriorization' of the affected gut with 'fistulization' is urged. Delore and de Girardier[7] believe that resection of the 'black pudding' through the receiving layer is often enough. Finally, Oughterson and Cheever[28] strongly advocate that every tumour of the intestinal tract, especially if it projects into the intestinal lumen, should be removed no matter how benign the character, unless there is some definite contra-indication, since it always carries with it the threat of intussusception.

## SUMMARY

1. A case is described of acute enteric intussusception in a man aged 52 years.

2. The cause of the intussusception was a submucous lipoma.

3. A search of the literature has shown that there are on record 242 cases of lipomata of the gastro-intestinal tract; 64 of these occurred in the small intestine.

4. Eighty cases of intussusception have been described as being due to lipomata, 34 being enteric in variety.

5. The case under consideration illustrates the modern theories of the mechanics of intussusception. These theories indicate that the tumour is not necessarily at the apex of the invagination.

6. Such a tumour is usually not discovered until it causes symptoms or until a laparotomy is performed for some condition of which it may or may not be the cause.

7. The treatment of intussusception due to a tumour is along the usual surgical lines, but search for a tumour especially in the proximal gut should be made in all cases of intussusception occurring after infancy, and, conversely, all benign tumours of the alimentary canal should be removed as being potential causes of intussusception.


I am grateful to Dr. R. P. Parker and Dr. R. A. Jackson for permission to to publish this case, and to Dr. Raymond Whitehead for the great care he has taken in preparing, mounting, and photographing the specimen.

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# CYSTS OF THE EXTERNAL CARTILAGE OF THE KNEE WITH EROSION OF THE HEAD OF THE TIBIA

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THE two following cases appear worthy of record :—

*Case* 1.—Mr. R., aged 49. Referred by Dr. Roberts, of Farnham, in February, 1930, for trouble in the left knee.

HISTORY.—Symptoms dated from a football injury in 1903, after which the knee swelled. The knee was weak for years and liable to go inwards and to give

FIG. 91.—*Case* 1. Cyst of the external cartilage of the knee eroding the head of the tibia of a man aged 49.

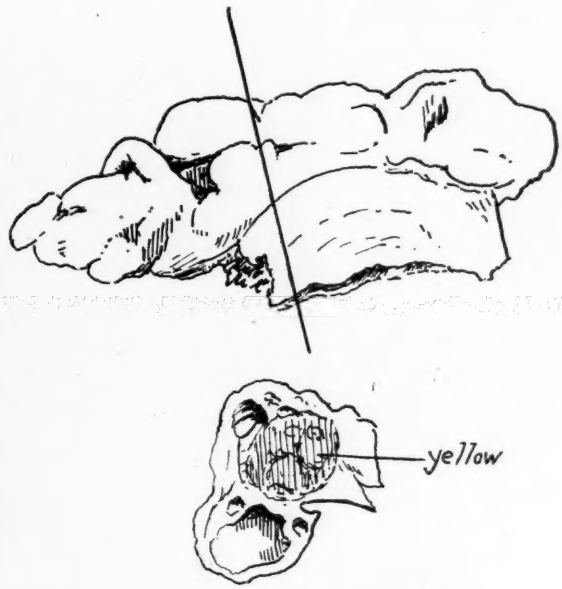


FIG. 92.—*Case* 1. Cyst of the external cartilage of the knee. (*Natural size.*)

sharp twinges of pain on the inner side ; never locked. It eventually got well and remained well for many years. The patient was able to play tennis but dared not jump. In 1925 he began to have pain on the outer side of the knee, the pain extending upwards and downwards on the outer side of the leg. There was a tender spot, and later a swelling appeared. The swelling is always present, but varies in size. Every six or eight weeks it gets worse, more swollen, and more tender. Increase of symptoms definitely due to excessive walking. When bad,

knee aches at night. It is stiff after sitting for long. The patient's doctor burst the swelling by firm pressure last year, and this relieved the patient at once.

ON EXAMINATION.—There was a typical elastic swelling, slightly tender, beneath and just in front of the external lateral ligament. Definite lipping of the bones. X-rays showed definite osteo-arthritic changes with a punched-out notch immediately below the outer margin of the tibia, between this and the head of the fibula (*Fig.* 91).

OPERATION (March 28, 1930—H. A. T. F.).—An unusually large multilocular cyst of the external cartilage was removed together with rather more than the anterior half of the external semilunar cartilage. There was a deep groove in the tibia from which the cystic mass was shelled, leaving bare bone, at the site shown in the X-ray. The groove was quite smooth and obviously due to pressure.

PATHOLOGICAL REPORT.—Examination of mass removed (*Fig.* 92) showed the mass of cysts extending right to the anterior extremity of the cartilage as well as beyond the line of the external lateral ligament. The centre of the tumour was bright yellow, with a fibrous network in it. The rest showed the usual fibrocystic appearance. The semilunar cartilage was split horizontally throughout its length. Microscopical section confirmed the diagnosis.

SUBSEQUENT HISTORY.—A recent report states that there has been no recurrence of the swelling or symptoms.

FIG 93.—*Case* 2. Cyst of the external cartilage of the knee eroding the head of the tibia of a man aged 36. There is also an old stellate fracture of the patella.

*Case* 2.—Mr. W. M., aged 36, had been troubled by his right knee for fifteen years and a swelling had been noticed for ten years. He attributed his symptoms to a fall which was followed six months later by a kick. The swelling was on the

outer side of the joint and was as large as a bantam's egg. He complained that it kept him awake at night. X-ray (*Fig.* 93) showed erosion of the outer tuberosity of the right tibia and an old stellate fracture of the patella.

OPERATION (May 25, 1932).—At the Royal Northern Hospital (E. I. Ll.). On incising the capsule a cystic mass was found to arise from the external semilunar cartilage. It measured 2¼ in. in an antero-posterior direction and was 1 in. wide.

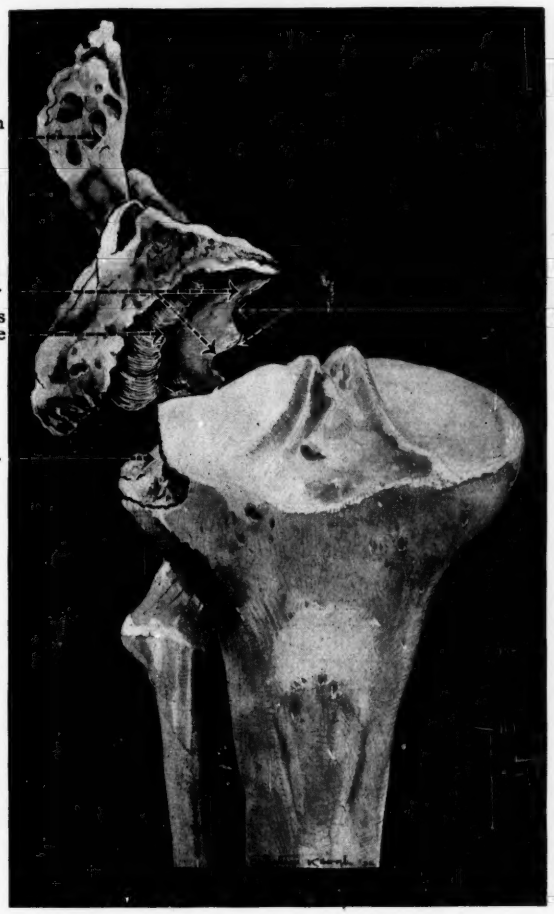
Upward prolongation of cystic mass
External cartilage
Portion of cystic mass which has eroded the tibia
Eroded area of tibia
Inner border of external cartilage

FIG. 94.—*Case* 2. Man aged 36. Composite drawing from X-ray and operative specimen. The tibia is seen from in front, and the external cartilage and cystic mass are shown as if lifted from the position which they occupied.

From its upper pole arose a process 2 in. long which passed upwards in the subcapsular tissue. The lower part of this large cyst had eroded the tibia and occupied a cavity in this bone ¼ in. deep and 1¼ in. long. The mass was removed with four-fifths of the external semilunar cartilage.

Mr. Charles Keogh, F.R.C.S., has kindly made a composite sketch from the X-ray and the cyst removed at operation. This clearly shows the relationship of the various parts (*Fig.* 94). The cyst was multilocular and contained gelatinous material. A microphotograph shows the usual appearances of such specimens (*Fig.* 95).

SUBSEQUENT HISTORY.—Eight months after operation the X-ray still showed the punched-out area in the outer tuberosity of the tibia, but the patient wrote recently that he is back at his old work as a French polisher and that the knee is

FIG. 95.—*Case* 2. Microphotograph of section. On the left of the field is a small cyst in the fibro-cartilage. It contains degenerate material and there is not a complete lining membrane. (× 40.)

"no disability except for a slight limp". Eight months later (i.e., a year and eight months after operation), he was walking normally and standing all day at his work

## SUMMARY

Two examples of cyst of the external cartilage of the knee-joint are described in which pressure of the cyst had produced changes in the outer tuberosity of the tibia which were obvious in the X-ray. Both cysts were unusually large: one had been present for nearly five years and the other for ten years.

# ADOLESCENT KYPHOSIS

By J. M. EDELSTEIN, Johannesburg

The general subject of kyphosis, apart from the well-recognized type following upon tuberculous infection of the vertebral column, is one which has received insufficient attention. Though such special types as rachitic round-back of small children, the round-back of senility, and the kyphosis which is sometimes a consequence of chronic ankylosing inflammation of the column have been known for many years, inadequate recognition has been given to that antero-posterior curvature of the spine which occurs in adolescence and which is a definite clinical and pathological entity. Some such cases must in the past have been regarded as those of healed Pott's disease, and others as the consequence of the relaxed round-back of school children, which cases were in turn vaguely ascribed to postural causes, to prolonged sitting at desks, etc. The spinal deformity that is here dealt with occurs in otherwise apparently healthy young boys and occasionally in girls, has no relation as far as we know to Pott's disease, and, resulting as it does in a fixed deformity of the column, is quite distinct from the round-back which is an error of posture and which can be corrected by the patient's efforts.

It is a remarkable fact that while American and Continental literature abounds in references to this subject, it is impossible to find a description of it in the British medical literature apart from a casual few lines in one or two surgical text-books.

Scheuermann,[1] of Copenhagen, in 1921 gave a full account of the condition and made suggestions as to its etiology. It was he who first drew attention to the significance of the epiphyses above and below the bodies of the vertebræ, and named the disease 'osteochondritis deformans juvenilis dorsi', though the lesion is now usually referred to as 'Scheuermann's disease'. He suggested that under certain circumstances changes occur in the epiphysial zone somewhat analogous to those which occur at the upper epiphysis of the femur in Legg-Perthes' disease, at the tibial apophysis in Schlatter's disease, etc.

The subject of adolescent kyphosis is of considerable importance. Not only is the condition disfiguring and in some cases disabling, but its differentiation from other forms of kyphosis, especially from that due to tuberculous disease, is obviously desirable. Furthermore, there is reason to believe that, were the condition better known and recognized in its early stages, appropriate therapeutic measures might be expected not only to stay the course of the disease, but to obviate the onset and progress of the serious deformity which is its consequence.

## CLINICAL MANIFESTATIONS

There is a striking difference in the incidence as between boys and girls. Of the 8 patients whose cases are here recorded, only 1 is a girl. Scheuermann's[1] cases showed a preponderance of 88½ per cent in males. Mau[2] described 13 cases, 3 of which were girls. Boerema,[3] describing 30 cases, includes only 1 female.

It is unfortunate that most of the cases present themselves for treatment when some, if not great, deformity already exists. The subject is usually an apparently healthy boy of between 12 and 17 years. Pain in the back is not a universal symptom, though some aching on exertion is usually complained of, and occasionally there is a good deal of pain. Very often, however, the first thing to draw attention to the condition is a deformity of great or less degree which the patient's parents or friends are usually the first to notice, the patient himself being as a rule unaware of the fact that he is assuming a curvature of the back, because from the front nothing abnormal may be seen. The curvature is almost always in the dorsal region of the spine, though some authors[4, 5] have described a lumbar spine involvement analogous to that in the thoracic region. The musculature of the back is uniformly excellent and the general health surprisingly good.

The curvature of the column is evident enough on examination (*see Figs.* 100, 101), and in addition to the kyphos there is sometimes a slight scoliosis. The back as a whole shows very little or no rigidity on active movement and the patient can move about freely, but the region of the kyphos is completely fixed and can be neither actively nor passively corrected; even by suspension and the use of the whole weight of the body one cannot noticeably diminish the kyphos, demonstrating that the deformity is a permanent one. The kyphos is not sharp, but gradually rounded and smooth, and is as a rule most prominent in the vicinity of the 7th to the 10th dorsal vertebræ. The ribs on each side are splayed out so that the thoracic cage acquires a dome-like character. Lumbar lordosis of more or less degree is often present to compensate for the curvature above.

In the vertebræ constituting the curved portion of the spine characteristic changes are to be seen on radiological examination. The radiographic appearances vary according to the stage of the disease, and as the interpretation of these appearances is bound up with the question of the normal ossification of the vertebral column, a brief consideration of the latter subject is necessary before the changes which follow upon the onset of the disease are discussed.

## NORMAL OSSIFICATION OF THE VERTEBRÆ AND ITS RADIOGRAPHIC FEATURES

At the 10th week of embryonic life three centres appear, one in each lateral arch and one in the centre of the cartilaginous body which is to form a vertebra. After the 6th year there is a period of quiescence of ossification.

The second period of more active growth in the adolescent stage finds its anatomical expression in the appearance of new centres of ossification, and at about the time of puberty newly acquired shadows are to be seen in the radiological picture of a normal spine. Above and below each body—first in the lower dorsal region and then extending up and down the column—there appear small triangular shadows in close proximity to their antero-superior and antero-inferior corners. From these triangular formations thin linear shadows extend backwards to become slightly broadened out again posteriorly (*Figs.* 96, 97). These appearances are those of the epiphysial plates—rings which are thickened peripherally and thin centrally. Before the appearance of the epiphyses the upper and lower surfaces of the body are convex; the plates are biconcave, and between them and the body are the transparent epiphysial cartilages. With this normal state of affairs the

intervertebral spaces are of course clear and the vertebral bodies are sharp and distinct in outline. It is only in a lateral view of the spine that the epiphyses are clearly seen, for in an antero-posterior view they are overshadowed by the articulating processes, etc.

FIG. 96.—X-ray photograph of the spine of a boy of 15 illustrating the normal appearances of the epiphysial plates above and below the vertebral bodies, as seen in lateral view.

These vertebral body epiphyses have been known for a long time, but their significance has not always been apparent, and as recently as 1920 the small triangular pieces at the anterior margins of the body were referred to in an important publication[6] as tuberculous sequestra.

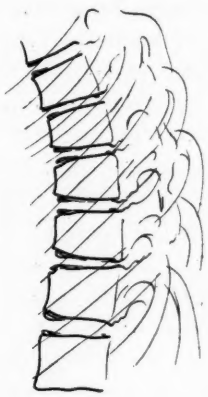

FIG. 97.—Diagrammatic representation of a normal spine at the age of 15, showing the epiphysial plates.

**Date of Appearance of the Epiphyses.**—The question of the date of the first appearance of these epiphyses is an important one and has been the subject of considerable debate. Gray[7] gives the age as 16 years. Scheuermann[1] says that 14 to 15 is the age of their first appearance. Mau[2] observes a difference in age of appearance according to sex, and gives the age for girls as 13 to 14 and for boys as 15 years. Personally, I have seen such epiphyses, or at any rate traces of such epiphysial formations, in skiagrams of several normal spines at as early an age as 12 to 13 years. This practically corresponds to the observations of Buchman,[8] who noted such epiphyses in spines of subjects as young as $11\frac{1}{2}$ years. There seems to be an individual variability, but I believe that some evidence of the newly developing centres can be observed in good X-ray negatives of most children of about 12 years of age.

**Union of the Epiphyses with the Body.**—The epiphyses unite with the vertebral bodies between the ages of 22 and 24 years, first in the cervical region, and last in the lumbar, and up to the latter age some sign of their partial or complete separation from the bodies is usually observed.

**Function of the Epiphyses.**—It is usually assumed that the epiphysial plates contribute to the growth in height and form of the vertebral body. Schmorl,[9] however, from his extensive studies of the development of the spinal column, has

denied that these are in fact growth epiphyses—a point of view which he utilizes in expounding an entirely different conception of the etiology of the disease from that which is usually accepted and to which further reference will be made.

## RADIOGRAPHIC CHANGES IN ADOLESCENT KYPHOSIS

Having discussed the ossification and the corresponding radiographic appearances of the normal spine at about the age of puberty, we may proceed to a consideration of the radiographic aspects of the affection. The pathological changes necessarily vary with the stage at which involvement of the spine is seen. These changes can be observed very clearly in a lateral view, and, when taken in the early phases of the disease, such an X-ray picture shows the epiphysial plates—especially anteriorly—rarefied, irregular, with their outlines frayed, and not sharp and triangular as is normal. The adjacent vertebral bodies show indistinctness of outline above and below, suggesting some abnormal process at the line of growth (*Figs.* 98, 99). This process is more marked anteriorly than posteriorly, with the result that the anterior surfaces of the vertebræ become shorter than the posterior, and even in the early stages some wedge-shaped deformation of the bodies is seen. The intervertebral spaces between the plates are no longer clear but cloudy and usually diminished in width.

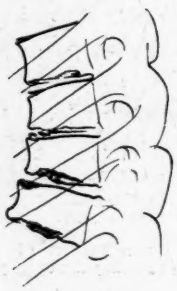

FIG. 98.—Diagrammatic representation of radiographic changes seen in an early case of adolescent kyphosis (*see Case* 1).

At a further stage there may appear actual fragmentation of the epiphysial plates with varying densities of the fragments. An appearance as of a 'field of ruins' is often seen, and it is difficult to decide whether these are derived from the body or from the bony epiphysis (*see Fig.* 99). The 'remains' appear as though pressed into the bodies, the latter showing more or less atrophy, especially at the apex of the kyphos.

The deforming and destroying phases may proceed very rapidly. As a rule, however, the process is of a very gradual nature and takes many years, during which time the deformity becomes progressively worse. As the disease runs its course, more distinctness becomes evident in the epiphysial plates, and they may even show a density above the normal. This is the reparative stage, which sets in gradually during the years in which the epiphysial line normally consolidates. There is a return of differentiation of the body outlines and their irregular contours become sharper and clearer. The onset of the third stage varies within fairly wide limits, which correspond to the variability of fusion time of the epiphyses, and, as in normal fusion, repair commences at the anterior parts of the body. With the conclusion of the growing age, the process of ossification being completed, the progress of the deformity is checked, but the wedging remains permanently, and, in the final adult picture of a case of adolescent kyphos, a dense appearance of the superior and inferior surfaces of the wedge-shaped bodies becomes evident.

The anterior corners of the affected bodies often take on a spur-like shape in the later stages—even one of my early cases showed this—and an appearance as of buttressing and bridging of the spondylitis deformans type becomes, in many cases, radiologically evident in the course of years.

The appearances above described are usually confined to two, three, or four vertebræ, most often the 7th to 10th dorsal being affected. The final deformation of the vertebral bodies varies with the severity of the initial process, and so gradations are seen of slight, medium, and severe wedging of the affected vertebræ, with corresponding degrees of spinal curvature.

Radiographically, the disease can therefore be traced from its beginnings in the years of puberty, continuing through the whole adolescent period, and only terminating at the time of cessation of normal growth.

## CASE REPORTS

*Case* 1.—F. C. B., schoolgirl, aged 14. For the past four months she had complained of some backache. The mother had noticed the child becoming increasingly round-shouldered during the last six months.

ON EXAMINATION.—Patient in robust health; more than the average height for her age. The back shows moderate kyphos in the lower thoracic region; there is slight tenderness on pressure over the spinous processes of 7th, 8th, and 9th dorsal vertebræ; no rigidity of the spine as a whole, but hyperflexion and hyperextension give rise to a little pain; musculature of back good. Nothing unusual in the past history; internal organs normal; kyphos is partially corrected on suspension.

*Radiological Examination* (*Fig.* 99).—Shows three dorsal vertebræ affected by a process typical of the earlier stages of the disease. This is best seen between the 8th and 9th, and 9th and 10th vertebræ. Here, in contrast to the clarity of the epiphysial plates of the vertebræ above, there is seen a hazy outline of the epiphyses and a blurring of the intervertebral spaces. The outlines of the inferior borders of the 8th, 9th, and 10th vertebræ are irregular, whilst in the inferior border of the 9th there appears to be a loss of substance, a small irregular piece remaining, however, which seems to belong to the vertebral body but is separated from it by a clear space. The 8th, 9th, and 10th vertebræ are already slightly but definitely wedge-shaped, and, what is very remarkable in a case of such short duration, the 10th vertebra appears saddle-shaped anteriorly with the antero-superior margin presenting a spur-like formation.

FIG. 99.—*Case* 1. X-ray illustrating characteristic appearances of an early case. Note the fragmentation of the epiphyses. Those of the 7th vertebra are unaffected, but the appearances at its lower border suggest the possible presence of nuclear hernia.

*Case* 2.—J. M., schoolboy, aged 14. Healthy, athletic, perfectly straight, until very severe attack of bronchopneumonia; after two months was discharged fully recovered, except for curvature of back noticed by nurses when he first got up; had never complained of pain in his back. Was treated by massage and exercises, but eventually was sent for investigation because Pott's disease was suspected, the deformity having become progressively worse. Complains now of backache on exertion.

ON EXAMINATION.—Patient is well-built but for marked curvature of the thoracic spine which embraces a higher level than usual; the curve appears to commence about the 4th dorsal vertebra, sloping gradually to its greatest prominence at the level of the 7th. Shoulders drooping; chest very flat anteriorly (*Fig.* 100); scapulæ very prominent; slight deviation

of spine to the left, most marked at 8th dorsal vertebra; whole back markedly arched. No lumbar lordosis, but the neck is forced forward, carrying the head prominently in front of the line of the body (*Fig.* 101). Cervical and shoulder movements normal; region of kyphos fixed and unalterable. No muscle rigidity. Movements of flexion, extension, and side bending free.

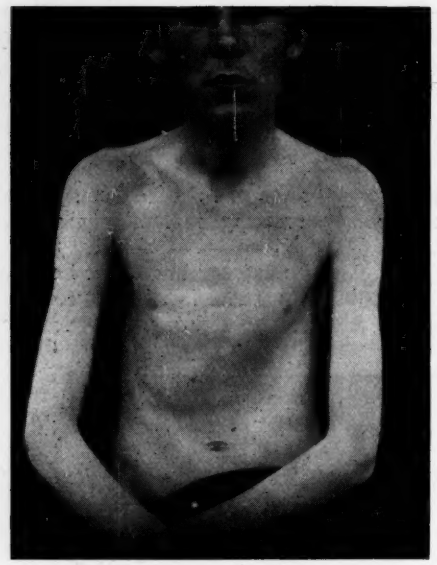

FIG. 100.—*Case* 2. Photograph of patient showing flattening of chest as seen from the front.

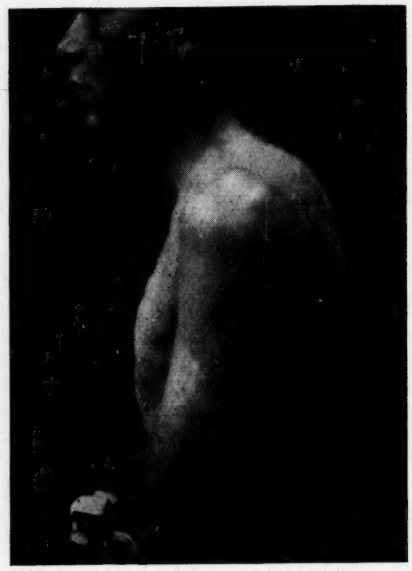

FIG. 101.—*Case* 2. Photograph of patient showing lateral view of deformity.

*Radiological Examination.*—Shows considerable blurring of the intervertebral spaces between the 6th and 7th, and 7th and 8th dorsal vertebræ. Some fragmentation of the epiphysial plates is seen anteriorly. Wedging of the vertebræ is a marked feature, whilst the borders show decalcification.

The case is of considerable interest in view of the history of onset either during or very soon after the attack of bronchopneumonia.

*Case* 3.—C. A., schoolboy, aged 15. Four months ago the parents noticed that he was becoming round-shouldered; has had no pain, but has lately felt disinclined to take part in sport; had been diagnosed by his doctor as a postural defect, but he is unable fully to correct the deformity. No previous illnesses.

ON EXAMINATION.—Well-built boy of normal height and weight; marked kyphos in lower dorsal area extending from 7th to 12th dorsal vertebræ. The curvature is to some extent, but by no means fully, correctable; hyperextension free; slight limitation of lateral movements and of flexion; compensatory lumbar lordosis; slight scoliosis; left lower ribs more prominent than right; slight tenderness on pressure over vertebral spines at kyphos.

*Radiological Examination* (*Fig.* 102).—Shows general blurring of the shadows of all the lower dorsal epiphyses; in fact, the anterior parts are practically unrecognizable. The margins of the vertebral bodies are everywhere uneven, and the contiguous surfaces of the 10th and 11th show considerable excavation anteriorly, with no trace whatever of the epiphyses; the intervertebral space between the 8th and the 9th is markedly narrowed. Appearances are suggestive of an extensive derangement of ossification.

*Case* 4.—D. S., male undergraduate, aged 17. Developed "a cold" accompanied by pain in the side of the chest; got up after three days, but continued to have intermittent pain in the chest. He noticed at this stage that he was becoming round-shouldered; doctor's

examination revealed a spinal curvature, and, Pott's disease being suspected, he was sent to Switzerland for six months; has been wearing a spinal support since his return. Still complains of occasional ache in the right side of the chest, especially when he leans back; no pain in the back except when he tries to straighten himself or walks much without his spinal support.

On Examination.—Patient robust, height 6 ft. Generalized dorsal curvature with maximum convexity at 8th and 9th vertebræ; slight scoliosis to left, most marked at 5th and 6th dorsal vertebræ, with slight compensatory curves to the right above and below. Front of chest not flattened; chest expansion good; musculature of back excellent; no limitation of movement.

Fig. 102.—*Case* 3. X-ray illustrating early stage. The epiphyses are practically invisible and body margins very irregular.

Fig. 103.—*Case* 4. X-ray illustrating involvement of five vertebræ with great deformation of their bodies and fragmentation of their epiphyses.

*Radiological Examination* (*Fig.* 103).—As many as five vertebræ are seen to be affected, viz., 6th to 10th dorsal. There are marked irregularities and variations in density at the superior and inferior surfaces of these vertebræ, especially of the 6th and 7th. Considerable fragmentation of the epiphyses is evident. The intervertebral spaces between the 6th and 7th, and 7th and 8th vertebræ are greatly diminished, and the corresponding bodies show much deformation.

*Case* 5.—T. H. S., male, chemist's assistant, aged 17. Left school and started work at age of 14½. Friends commented on his becoming round-shouldered, and since then has been getting gradually worse; has never felt pain or the slightest discomfort; no past illnesses except bronchitis in infancy; no history of injury.

On Examination.—Patient is rather thin, but otherwise of good build; height 5 ft. 6 in. A gradual extensive curvature in the thoracic region with its greatest prominence at 8th and 9th dorsal vertebræ; thorax dome-shaped; scapulæ with lateral and posterior parts of thoracic cage projected prominently backwards. Musculature of back extremely well

developed; some lumbar lordosis present; flexion and side-to-side movement normal; no muscle spasm; hyperextension at the lumbar spine easily performed. Region of kyphos completely immobile and resists all attempts at correction.

*Radiological Examination* (*Fig.* 104).—The epiphysial plates above and below each vertebral body are clear and triangular in the upper part of picture, but those between the 7th and 8th, and especially between the 8th and 9th, show blurring of their shadows. The plates appear to be compressed and flattened out. The interval between the 9th and 10th thoracic vertebræ is of interest because while no epiphyses can be seen here, the whole of the upper margin of the 10th shows serration. The 7th and 8th bodies are wedge-shaped.

FIG. 104. — *Case* 5. Illustrating contrast between normal and affected epiphyses, the latter showing increasing degree of involvement from above downwards.

FIG. 105.—*Case* 6. X-ray illustrating marked wedge-formation of vertebræ and spur-like appearances at anterior margins.

*Case* 6.—W. H. M., shoemaker, aged 19. Since the age of 14 he was often upbraided by his schoolmaster for not standing upright. Found it impossible to hold himself erect for more than an hour, and gradually became more and more round-shouldered. Never had pain, but sought advice only in view of his ungainly appearance; not handicapped in any way; plays football and other games without difficulty.

ON EXAMINATION.—Excellent physique, height 6 ft. Marked rounded kyphos, with whole thorax domed backwards and prominence of 7th, 8th, and 9th dorsal vertebræ; marked lordosis; also pronounced forward position of upper dorsal and cervical vertebræ; movements normal except flexion, which, though good, does not enable him to touch his toes.

*Radiological Examination* (*Fig.* 105).—Shows considerable wedging of the 8th, 9th, and 10th thoracic vertebræ, the superior and inferior margins of which present an appearance as though the epiphyses, misshapen but still visible, had been pressed into the bodies anteriorly. The upper border of the 10th vertebra shows considerable excavation. A notable feature is the spur-like formation of the vertebral margins anteriorly.

*Case* 7.—N. C., bricklayer's assistant, aged 18. At the age of 14 his father noticed that his back was deformed; family doctor made a diagnosis of "consumption of the spine"; was sent to convalescent home for some months; more recently has undergone gymnastic exercises and massage without effect.

ON EXAMINATION.—Rather thin and anæmic, height 4 ft. 10 in.; has no pain, but is much concerned by the deformity. Marked kyphos in lower dorsal region, most prominent at 7th, 8th, and 9th dorsal vertebræ; thoracic cage very prominent on either side; some compensatory lordosis; chest expansion poor. Movements of back remarkably good; can easily touch toes; region of deformity quite rigid.

*Radiological Examination.*—Shows considerable wedging of the 8th, 9th, and 10th dorsal vertebræ; there is some irregularity and increased density of their superior and inferior borders. No epiphyses are to be seen, and the appearances suggest the healed stage of the disease.

FIG. 106.—*Case* 8. X-ray illustrating healed stage, with more or less reconstituted vertebral margins.

*Case* 8.—C. G., baker's assistant, aged 24. Went to doctor a year ago because of pain in the back; has never had pain before, nor has he worried about his spinal deformity, of which he has been aware for the past seven years; has always been athletic and has only been hampered in his duties recently because of pain.

ON EXAMINATION.—Muscular and well-built, except for moderate curvature in region of 8th, 9th, and 10th dorsal vertebræ. Movements slightly limited; kyphos is fixed; slight lordosis; some tenderness over kyphos; wears spinal support, which relieves his pain.

*Radiological Examination* (*Fig.* 106).—Shows wedging of 8th, 9th, and 10th dorsal vertebræ; their margins are reconstituted but for slight irregularities and variations in density; the intervertebral spaces are diminished; the epiphyses are united; there is no spur-like formation. The pain at present complained of suggests commencing arthritis.

## ETIOLOGICAL CONSIDERATIONS

The question of the etiology of adolescent kyphosis, though of the greatest interest, is shrouded in much obscurity.

Is the deformity the consequence of interference with the normal development of those structures responsible for the proper growth in height and shape of the vertebræ, or is the effect on these structures merely secondary to a kyphotic deformity from other causes?

Change of structure in the early or florid stage is obviously not simply the result of compression, because change of structure can be seen to precede change of form. Buchman[8] mentions two cases without spinal deformity which showed a radiographic involvement of the spine similar to other cases with deformity typical if the disease—a discovery which convinced him that the epiphysial involvement is primary.

**Derangement of Epiphysial Development.**—From many points of view the theory of an interference with the normal development of the epiphysial plates as a causative factor has much in its favour, though what is responsible for such interference is far from clear. Histological sections from cases of actual disease are of course not available, as the condition is non-fatal. There can be said to be as little agreement as to the cause of this interference in the case of the spine as there is regarding the other epiphysial disturbances such as

characterize Legg-Perthes' disease of the hip, Köhler's disease of the tarsal scaphoid, etc.

On the assumption of a defect in epiphysial development being responsible for the supervening kyphosis, the latter would further disturb, by pressure, the growth which has already been delayed by the original disease. A vicious circle thus occurs which ceases at the moment when re-calcification supervenes in spite of all, and the deviation becomes fixed.

**Theory of Infection.**—There is no clinical justification for believing that the condition is an inflammatory lesion brought about by infection. Axhausen's suggestion in regard to the etiology of other epiphysial affections—that emboli of avirulent organisms interfere with the circulation and cause a necrosis in the epiphysial areas—if true at all in such cases, can hardly apply to the spine, as the process extends to several vertebræ at the same time. It is far-fetched to assume that several epiphysial rings should fall victims to an infection at the identical time.

**Theory of Increased Load and Diminished Capacity.**—The onset of the disease at the age of puberty, and especially in boys, when the body framework is called upon to withstand the demands of greater activity at a period of active bone generation, suggests the possibility of mechanical strain on such actively growing areas as a factor in the etiology. Mau[2] especially stresses the importance of mechanical strain on, and diminished capacity of, the actively growing spine.

1. *Functional Trauma.*—Most writers on the subject emphasize the special incidence of the disease in agricultural labourers and in others who are subjected to hard manual labour, excessive lifting, etc. It is suggested that an increased static demand may result in marked changes at the line of growth of those vertebral bodies which mechanically are subject to greatest strain, and it is certainly true that the 8th, 9th, and 10th vertebræ might be especially vulnerable in that regard, since, owing to the normal thoracic convexity, they are situated far behind the line of gravity. In his most recent article on the subject Scheuermann[10] gives it as his opinion that "the ossifying process is affected either by an isolated severe traumatism or by a series of occasional traumatic lesions. There is then a temporary overloading which produces in the anterior parts of the vertebræ, where the effect of the lesion is greatest, a temporary arrest of ossification."

In a series of experiments on rats whose tails he had kept in a state of extreme flexion by stitching them to the abdominal walls for long periods of time, Mau[11] observed alterations in the epiphysial cartilages amounting to atrophy and cessation of endochondral bone formation. It is debatable whether one can apply to the disease under discussion, in adolescent human beings, such a conception based on extreme compression of vertebræ in rats.

In none of my cases has there been special evidence of undue occupational strain, and I doubt whether this factor plays the all-important role ascribed to it. All the cases under my observation had a very well-developed musculature, and *Case* 2 is particularly instructive in this connection, as the deformity was definitely first observed after a lengthy period of recumbency.

2. *Diminished Capacity of the Growing Spine.*—This is a factor not easy to evaluate. Whilst it is true that during the period of active growth the strength of bone is diminished, we do not see in *normal* growing bone any inability on the part of the tissues to adapt themselves to increased functional requirements. It is therefore doubtful whether the physiological weakness which goes with rapid

growth is a very important cause of diminishing the capacity of the column to bear weight. Nor can so-called 'late rickets' as a weakening factor be incriminated, since, apart from the fact that there are no other deformities present, recent researches[12] into the blood chemistry of the various types of the osteochondritides, which included 19 cases of vertebral epiphysitis, have shown that the calcium and phosphorous contents of the blood serum fall within normal limits.

**Schmorl's Theory of Nuclear Hernia.**—The supposition that the *primary* cause of adolescent kyphosis is some disturbance of epiphysial development is seriously challenged by Schmorl.[13–15] According to him, the views hitherto held on the function of the epiphysial plates are erroneous—that rather than being concerned with the growth of the vertebral body itself, they merely form, after cessation of endochondral growth, the circumferential edge for fixation of the intervertebral discs. The vertebral bodies, covered on their intervertebral aspects by cartilaginous tissue—the cartilage plates—develop like short bones from the subchondral zone. The cartilage plates are set directly on the spongy cancellous tissue at the upper and lower aspects of the vertebral body. The two structures are called upon to withstand the pressure exerted by the nucleus pulposus of the intervertebral disc, which is confined between the two cartilage-covered vertebræ. There is no layer of compact bone, and even the cartilage plates are present only over the central parts of the body. It is to the peculiar elastic properties of the nucleus pulposus of the disc that we must look, according to Schmorl, for an explanation of disturbances in the osteogenetic zone of the vertebral body. The rôle presumably played by the nucleus pulposus in the etiology of spinal deformity and disease has received a great deal of attention, and Beadle[16] has recently published an extensive account of the researches on the subject.

Prolapse of the nucleus pulposus may, according to Schmorl, occur as the result of congenital defect of the cartilage plate. Such possible disturbances of development were suggested by his examination of 4000 spines amongst which he found, in quite young children, evidence of nuclear prolapse in the form of "cartilaginous knots" in the spongiosa of the vertebral body. Such "cartilaginous knots" are regarded as lying within the range of normality and are said to assume a pathological character only when, under the influence of great functional demand, there occur other changes, such as further tearings of the cartilage plate with further herniation of nuclear tissue into the spongiosa.

Having demonstrated such nuclear prolapses even in children, Schmorl assumes a congenital predisposition to the development of adolescent kyphosis. The deformity occurs only during adolescence because, from overloading of a potentially weakened spine, a disproportion exists between the resistance of the intervertebral plates and the weight. Trauma pure and simple, without the presence of congenital defect, may also result in herniation of the nucleus pulposus by tearing a normal cartilage plate.

The pathological sequence of such nuclear prolapse is conceived as follows. Where, with overloading or trauma, there is nuclear prolapse into the spongiosa, there the process of ossification in the *subchondral zone* is distributed, so that there is an interference with the growth in length of the vertebral body, since such growth depends upon proliferation of the spongy tissue of the body itself. As a consequence of the prolapse of the disc, however, there is a reactive new formation of bone, and, near the upper and lower surfaces of the body, irregularities of

ossification occur which account for the serrated margins of the body and for the presence of areas of condensed bone around the periphery, both of which can be seen radiologically. This disturbance holds back the growth of the epiphysial ring and may even distort it, but the marginal irregularities, according to Schmorl, are not due to an epiphysial involvement as such, but to the effect on the *bodies* of the prolapse of the nucleus pulposus.

There has been considerable support for the views of Schmorl on the etiology of adolescent kyphosis, though they are based entirely on post-mortem evidence and not at all on clinical data. It is true that the inferences are drawn from a series of spines in not one of which was there post-mortem evidence of epiphysial disturbance, and that the series included six young subjects with kyphosis, who had died between the ages of 16 and 24. The dates of development of the kyphoses are, however, not given, as clinical histories were not available. It is thus quite uncertain whether the six cases of kyphosis referred to by Schmorl belonged to the category of 'adolescent kyphosis'. Furthermore, reconstitution of affected epiphyses after the subsidence of the early stages of the disease is not only a possibility, but is definitely suggested by the radiological appearances in the late stages.

There are, indeed, many clinical and radiological aspects of adolescent kyphosis which remain unexplained by Schmorl's theories. The characteristic blurred outlines of the *anterior* parts of the vertebræ in the 'florid' stage of the disease are difficult to explain on such a conception, especially in those cases with comparatively rapid onset of the kyphosis. It is also not clear how the typical cycle of changes lasting over several years can be regarded as a process in which the epiphyses play but a secondary part. Neither is that part of the theory which presupposes a traumatic factor borne out by the histories of the cases under my observation.

As to the radiological appearances, one would expect that evidence of such nuclear prolapse would be forthcoming in all cases of adolescent kyphosis. With the possible exception of *Case* 1, where the appearance of the *unaffected* body of the 7th vertebra is suggestive (*see Fig.* 99), I have failed to satisfy myself that such exists, either in my own comparatively small series of cases or in that of a larger group of X-ray negatives typical of the condition which I recently had the opportunity of examining at the Clinic of Professor Haas in Vienna.

It is of interest in this connection that Calvé and Galland,[17] who subscribe to Schmorl's views, yet find it necessary to invoke a purely epiphysial lesion independent of nuclear prolapse as an explanation for at least 3 of their series of 26 cases. A remarkable case is recorded by them which, on first observation at the age of 15 years, showed a kyphosis with "epiphysitis and sub-epiphysial rarefaction", and which *two years later* showed radiological evidence of "very small nuclear hernias". Six years later, while the epiphyses were united and the vertebræ wedge-shaped, disc hernias were found to be numerous and prominent.

That nuclear prolapse occurs cannot be denied, many radiological observations of such lesions having been reported, but whether such prolapses have any bearing on the etiology of adolescent kyphosis is by no means proven. Radiological evidence of nuclear prolapse in cases of such deformity may be purely incidental.

**Other Suggested Causes.**—That the deformity has anything to do with muscular asthenia can be dismissed by the fact that the muscle development is always good.

Endocrinal disturbance affecting normal bone growth has been suggested here as in other epiphysial derangements, but there is at least no naked-eye evidence to justify such an assumption.

The idea of circulatory disturbances at the line of growth from functional trauma, leading to vasospasm, ischæmia, and aseptic necrosis, fails to explain why the epiphyses of some children are so much more vulnerable than others.

## CONCLUSIONS AS TO ETIOLOGY

If one discards the congenital basis of Schmorl's conception of the etiology of adolescent kyphosis, one is forced to the conclusion that a developmental disturbance arises *de novo* during the years of puberty. What the nature of that disturbance is, and what its immediate cause, awaits elucidation, but no doubt the question is bound up with the general problem of the etiology of osteochondritis in young subjects. It is possible that a nutritional defect, inimical to normal epiphysial development, will prove to be the key to the problem of disturbances in the vertebral column as well as to those in other areas of epiphysial growth. We have in rickets an example of an error of assimilation, or of nutrition, or of both, which affects growing bone so profoundly that distortion comes with ordinary weight-bearing. There are many other biochemical factors besides those of the assimilation of calcium and phosphorus about which, in relation to growing bone, we understand too little.

One cannot but feel that all the etiological possibilities previously discussed are in the final analysis at most of secondary importance only, and that one has to seek for some hitherto undetermined biochemical disturbance to explain why the epiphyses of some children fall victims to circumstances which in others have no effect. It is probable that the only significance of the much-vaunted factor of " multiple small traumata " is that of a localizing or, at most, an aggravating agency.

In connection with the general subject of epiphysial growth disturbance in children, another lesion of the spine which occurs in the 2 to 7 year age-period may be mentioned. Described by Calvé,[18, 19] under the name of " osteochondritis of the vertebral body ", it bears certain analogies to the disease which is the subject of this article, and results in an interference with the development of the *primary* epiphysis for the body centrum. The affection, one example of which has come to my notice, leads to flattening and sclerosis of the affected vertebra and to the development of a kyphos. The analogy between this disease and adolescent kyphosis is emphasized by the fact that in both cases the age of onset is the period of most active growth for the area involved. May there not be also some analogous nutritional factor in both diseases ?

## TREATMENT

It is not an easy matter to formulate the treatment of a condition the etiology of which is still so obscure. It is, however, necessary to stress the importance of early recognition of the disease. The onset of even the slightest kyphosis, or of what is popularly known as ' round shoulders ', in a subject at the age of puberty should demand a radiological examination at the earliest possible moment. Prophylactic measures in the early stages should do much to minimize deformity. It is

essential, as was carried out in *Cases* 1 and 2, that in such comparatively early cases complete recumbency should be adopted in order that weight-bearing on the affected column may be reduced to a minimum. In recumbency the ventral parts of the whole column are pulled apart and further compression of the bodies and their epiphyses is lessened. This effect can be heightened if a suitable frame is provided to keep the spine hyperextended. Traction to the legs and head, with elevation of the upper end of the bed, is of great advantage. After a minimum period of three months on the frame, and longer if radiological control demands it, the patient may be let up in a plaster-of-Paris jacket or a leather support. The latter appliance, worn for several years after, is of value in preventing the increase of residual deformity, which unfortunately occurs to some extent, in spite of most careful treatment. Pain is most certainly relieved by the wearing of a posterior support.

It is possible that earlier radiological examination of youths who complain of 'backache' may reveal the condition before the appearance of kyphosis. This, of course, is not a simple matter because so many of the cases have few or no symptoms in the early stages.

It is very doubtful whether for those cases with deformity of considerable duration much can be accomplished. Mobilizing measures such as gymnastic exercises, massage, etc., are of negligible value at a stage when fixed rigidity has been present during several years. One's own experience is that orthopædic gymnastics have not the slightest effect on the deformity in the very late cases, and it is of course obvious that in the earlier stages such measures are definitely contra-indicated. There are, however, intermediate stages where the deformity has not yet reached its maximum and where measures to prevent the progress of such deformity are called for. Plaster-of-Paris jackets, put on in as much hyperextension of the spine as it is possible to obtain, are to be applied and re-applied at intervals, until such time as the deformity shows no tendency to progress. As a precautionary measure a posterior spinal support may be used later.

Treatment on the whole cannot be said to be satisfactory in its results, and that is perhaps to be expected, in view of the present state of our knowledge with regard to the etiology of the lesion which is responsible for the deformity.

## SUMMARY

1. A kyphotic deformity may occur in adolescents resulting from disturbances of vertebral ossification.

2. The ossification of the column and the deviations from the normal consequent upon the onset of the growth disturbances are described.

3. The lesion results in wedge-shaped deformation of several vertebral bodies. The kyphosis which this gives rise to, being sometimes unaccompanied by pain, may at first pass unnoticed. Movements of the spine are but little affected, but disfigurement is progressive.

4. Eight cases are reported illustrating various phases of the disease, and their radiological features are described.

5. A critical survey of the etiology is presented and the conclusion is drawn that most, if not all, the etiological factors usually cited to explain this and other epiphysial disturbances of a similar nature are of secondary significance only.

6. The tentative suggestion is made that some biochemical disturbance, based possibly on an error of nutrition or of assimilation, is at the root of such epiphysial affections.

7. The lines of treatment are indicated and special emphasis is laid on the importance of early recognition of the disease.

I desire to acknowledge my indebtedness to the orthopædic surgeons of Liverpool for permission to include some of their cases in the group which is the basis of this article.

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# ENTEROGENOUS CYSTS

By W. E. A. HUGHES-JONES

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THE following are the notes of a patient who was under the care of Mr. A. E. Coates at the Melbourne Hospital.

A. McA., male, aged 32 years. For many years there had been pain about two hours after meals, intermittent attacks of vomiting, each lasting for four weeks, and remissions lasting about six weeks. During the attacks vomiting would occur about three times a week, without warning half-way through a meal. A barium meal examination suggested the presence of a duodenal ulcer. For twelve hours there had been severe epigastric pain of sudden onset. Vomiting occurred once; there was much nausea and retching, and the bowels had not acted.

ON EXAMINATION.—The temperature was 95·6°, pulse-rate 64, and the blood-pressure, systolic 92 mm., diastolic 54 mm. The abdomen was not distended, was held immobile during respiration, and there was generalized rigidity, most marked in the upper abdomen, especially on the right side. Acute tenderness was elicited midway between the xiphisternum and the umbilicus, and the area of liver dullness seemed diminished. A diagnosis of perforated duodenal ulcer was made.

OPERATION.—At operation there was no evidence of gastric or duodenal ulceration. Some blood-stained fluid escaped, the upper loop of the jejunum was distended, and the lower ileum was collapsed. The small bowel was followed proximally till near the upper part of the jejunum there was found a strangulated cyst in the left side of the mesentery, about three-quarters of the size of a tennis ball. The pedicle was ligated and the cyst removed The wall of the cyst was identical in structure with that of the adjoining small intestine

## ETIOLOGY OF ENTEROGENOUS CYSTS

The view most frequently accepted is that enterogenous cysts arise from diverticula of the intestine of the embryo. Another theory is that epithelium becomes detached from the embryonic intestine at a very early age and develops into a cyst. Each of these conceptions will be discussed.

**The Theory of Origin from Diverticula.—**

1. Those who believe that cysts arise from diverticula have no difficulty in demonstrating many such diverticula in the intestine of the embryo. Reference is often made to the work of Lewis and Thyng, and although such diverticula as they described on the free border of the developing intestine may become isolated from the bowel and form cysts, it is unlikely that this occurs with any degree of frequency.

Another possible origin is found in the irregular epithelial growth which occurs at the stage when the intestinal lumen of the embryo becomes obliterated, for small hollow stalks of cells bud into the surrounding mesenchyme and have been found cut off from the intestine in some instances.

In a very careful investigation of hyperplasia of the mucous membrane of the intestine, Lauche demonstrated that sometimes intestinal glands transgressed the muscularis mucosæ of the stomach, ileum, and jejunum, and in some instances

he found in the submucosa small cysts which appeared to arise by an isolation of the glands from the intestinal mucosa. Stohr, Florence, and Sabin have described similar glands in the embryos of domestic animals and of man occurring in the lymphoid follicles of the ileocæcal region.

From such evidence it appears that there exist in the *embryo* numerous diverticula from which enterogenous cysts may develop. It is possible that some cysts do arise in this way, but such is a matter of speculation, and complete proof is almost unattainable.

2. It is very unlikely that enterogenous cysts and non-vitelline diverticula of the *adult* small intestine have a common origin, for their distribution differs so widely. Diverticula occur most frequently in the jejunum, especially in its upper coils, whereas enterogenous cysts are very uncommon in the jejunum and are found in the ileum, especially near the ileocæcal junction. The age incidence of diverticula and of enterogenous cysts differ widely, since the former are found in the adult and the latter usually in infancy and in childhood.

3. Another theory of the development of enterogenous cysts is worthy of serious consideration. Gfeller regards enterogenous cysts as arising from an unobliterated vitello-intestinal duct, either by separation of a part of the intestinal *Anlage* or by germinal displacement. Edwards and Dukes think this is unquestionably the most likely hypothesis, but it seems to the author that the widespread distribution of the cysts is the greatest argument against the application of this theory to all cases. There are certainly some instances where it is impossible to gainsay that a cyst in the antimesenteric border of the bowel or in the mesentery may be derived from the vitelline tract. Indeed, most of the antimesenteric cysts probably arise in this way because, for reasons not understood, enterogenous cysts of non-vitelline origin are entirely situated toward the mesentery (perhaps the association of germinal displacement with blood-vessels provides the clue). But Meckel's diverticula and associated remnants are in a fairly fixed position, and the greatest departures from this position which are known to the author are, at one extreme, at the appendix (Crymble's case), and, at the other, midway in the length of the small bowel. It is difficult to believe that thoracic, duodenal, and ileocæcal enterogenous cysts have their origin in the vitelline duct.

The position is that enterogenous cysts appear to arise from any portion of the embryonic alimentary tract, more frequently in certain sections, and that cysts resembling enterogenous cysts in structure and characteristics arise from the vitelline tract. The latter equally merit the name 'enterogenous cyst', but they must necessarily be of limited distribution.

**The Sequestration Theory.**—A very careful search is necessary in order to find evidences of the early stages of epithelial sequestration, and very few examples have been described; but this is not a bar to an etiological relationship to enterogenous cysts, since these cysts are themselves very uncommon.

The author made serial sections of the intestines of a number of human embryos, and in two embryos evidence of epithelial sequestration was found. Illustrations of these sections are presented (*Figs.* 107, 108). *Fig.* 107 is from a 65-mm. embryo; the small cyst extended through three sections, the epithelium appeared undifferentiated, and in this resembled the epithelium of the adjoining intestine. The section did not include any Wolffian remnants. *Fig.* 108 is from an embryo of 170 mm. The small cyst was lined by epithelium resembling that

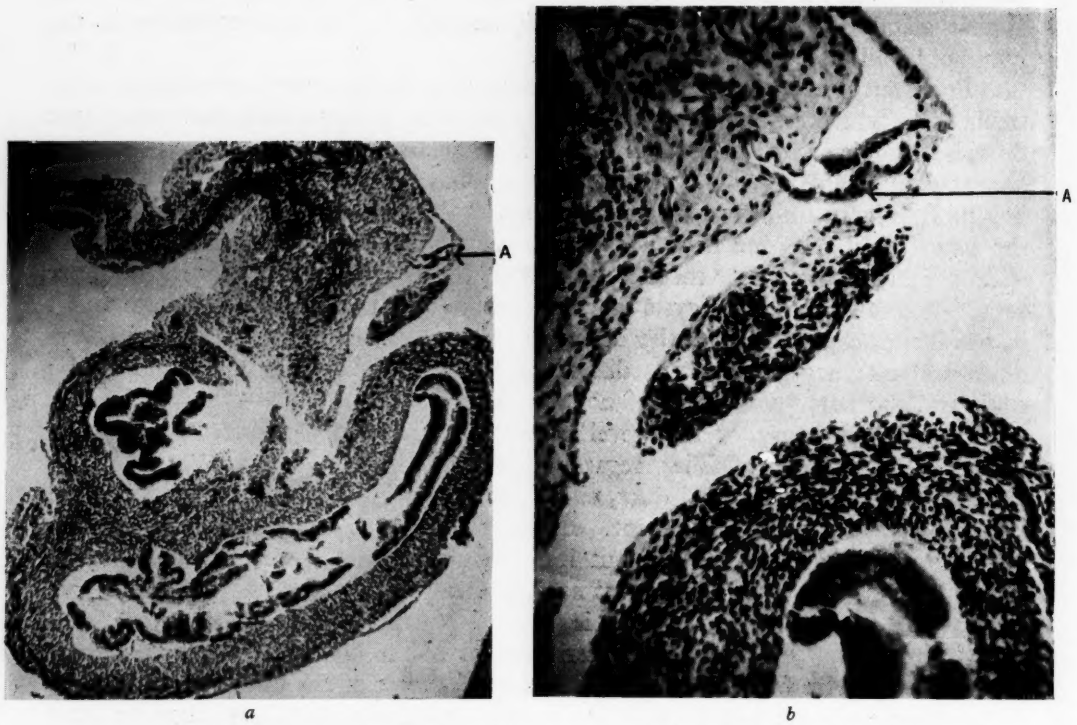


FIG. 107.—Transverse section of the small intestine of an embryo of 65 mm. An isolated group of cells is seen at (A), and, when traced through serial sections, these cells were seen to form a closed cavity. *a*, Low power (× 37); *b*, Higher power (× 120).

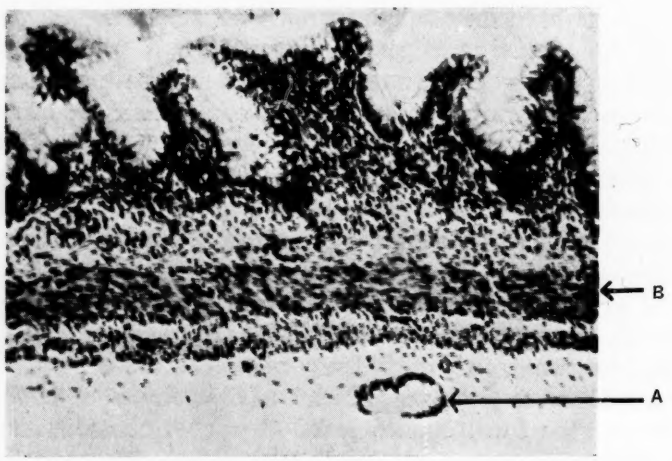


FIG. 108.—Transverse section of the small intestine of an embryo of 170 mm. At A is seen an island of epithelium lying outside the muscularis of the intestine (B). The epithelium resembled that of the adjoining intestine. (× 37.)

of the bowel. Such observations prove nothing, but they demonstrate a possible source of origin for enterogenous cysts.

These small islets of epithelium must have been detached from the intestinal mucosa before the circular musculature was formed. This muscle appears in the duodenum in a 12·5-mm. embryo and spreads along the intestine until it is present in the ileocæcal region in the 22·8-mm. embryo. Thus the sequestration of the epithelium probably occurs before the embryo reaches the 23-mm. stage. Sequestration of buds of intestinal epithelium may occur in the submucosa over a much longer period, for the muscularis mucosæ is not differentiated until the embryo measures 187 mm.

It is assumed that this isolated epithelium gains a muscle coat in the same way as the intestine by a condensation of the hitherto undifferentiated mesenchyme which surrounds the intestine.

Several workers have made observations which resemble those described except that the cysts they report were confined to the duodenum. In a pig embryo of 20 mm. Lewis and Thyng described a cyst which had become detached from the intestine in the lower duodenal region. Johnson found among the duodenal villi of a seven-month embryo several small cysts which were distended with mucus and were separated from the surface epithelium by a thin layer of connective tissue.

## DESCRIPTION OF THE ENTEROGENOUS CYST

The wall consists of three layers, muscular, submucous, and epithelial. The muscular layer usually consists of two strata lying at right angles to one another, but occasionally a thin single layer is found. It is this coat which causes the cyst to resemble the intestine so closely. The submucosa is a thin connective-tissue layer. Epithelium may form a complete lining, or be present in limited patches, or even be completely absent, but in this last event epithelial cells are usually found in the contents of the cyst. The epithelium forms a thin smooth layer, occasionally possesses glands, and is most frequently columnar or low cuboidal, but it may undergo transition to ciliated or stratified epithelium.

The fluid from the cyst is usually pale, clear, or straw-coloured, containing much albumin; the reaction is alkaline and the specific gravity is 1015. The fluid is occasionally distinctly mucoid. Microscopic examination sometimes reveals blood, cell débris, and cholesterin.

**The Site of the Cysts.**—An idea of the relative frequency in different portions of the small and large bowel is gained from a tabular summary of the 55 cases :—

| | Cases |
|---|---|
| Duodenum | 3 |
| Jejunum | 4 |
| Ileum (excepting last 4 in.) | 16 |
| Last 4 in. of ileum, Ileocæcal valve, Ileocolic angle, Cæcum, First 2 in. of ascending colon | 31 |
| Transverse colon | 1 |

Of the 16 cases included as occurring in the ileum above the last 4 in., some may really belong to the lower 4 in., but an opinion could not be formed on the

published data. Even so, the frequency of the incidence in the ileocæcal region is worthy of note in contrast to the rarity of the cysts in the colon beyond the first few inches of the ascending colon and their infrequent occurrence in the duodenum and jejunum. The incidence in these latter organs does not appear to exceed that of enterogenous cysts in association with the trachea, œsophagus, or even the stomach.

FIG. 109.—The terminal ileum and cæcum of a child. Immediately above the ileocæcal valve two enterogenous cysts are to be seen, one in the submucosa obliterating the lumen of the intestine, and the other, deep to the serosa, projecting on the surface of the ileum. (*Reproduced by the courtesy of the Curator of University College Hospital Museum.*)

The distribution in the ileocæcal region is worthy of analysis :—

| | Cases |
|---|---|
| Ileum within 4 in. of ileocæcal valve .. | 12 |
| Ileocæcal valve .. .. .. | 3 |
| Ileocæcal and ileocolic angle .. .. | 3 |
| Cæcum .. .. .. .. | 10 |
| Ascending colon (first 2 in.) .. .. | 3 |

**Position of the Cyst in Relation to the Wall of the Bowel.**—Details of this are mentioned or can be deduced in 45 instances. The cyst may be: (1) In the submucosa; (2) In the muscle layers; (3) Subperitoneal; (4) In the mesentery of the bowel, either close to or some distance from the intestine. (*Figs.* 109, 110.)

The position of cysts in the wall of the jejunum and ileum differs from that of cysts in the ileocæcal region.

| | | Cases |
|---|---|---|
| In the jejunum the positions were .. .. .. | Intermuscular .. | 1 |
| | Mesenteric .. | 3 |
| In the ileum (excepting the last 4 in.) .. .. | Submucous .. | 2 |
| | Intermuscular .. | 3 |
| | Mesenteric .. | 9 |

That is, in these cases the mesenteric distribution was predominant.

In the ileocaecal region the incidence is greatest in the submucosa, and many cysts occur in the muscle layer.

| | | Cases |
|---|---|---|
| Ileum (last 4 in.) .. .. .. | Submucous | 5 |
| | Intermuscular | 4 |
| | Mesenteric | 1 |
| Ileocæcal valve .. .. .. | Submucous | 2 |
| | Subperitoneal | 1 |
| Cæcum .. .. .. .. | Submucous | 4 |
| | Intermuscular | 2 |
| Ascending colon .. .. .. | Submucous | 3 |
| Ileocæcal and ileocolic angle .. .. | Subperitoneal | 3 (i.e., mesenteric) |

Another feature worthy of note is that, of the cysts in any part of the ileum, 10 were on the mesenteric side of the bowel and 5 were antimesenteric (these data were supplied only in a limited number of cases). The antimesenteric cysts are believed to originate in the vitello-intestinal duct.

**Age when Discovered.**—As a rule enterogenous cysts are found only when they cause severe mechanical disturbances in the intestine. A study of the age

FIG. 110.—A specimen of great interest. Closely attached to the transverse colon is a thick-walled enterogenous cyst, A diverticulum, resembling in all respects a diverticulum of the colon, is present in the wall of the cyst. (*Reproduced from St. Thomas's Hospital Museum by kind permission of the Curator.*)

at which such complications occur reveals features of interest, and a brief analysis is set out :—

| ORGAN | AGE WHEN CYST WAS DISCOVERED |
|---|---|
| Duodenum .. .. | Close to birth |
| Jejunum .. .. | 2 in the first week of life<br>1 at 6 years<br>1 at 32 years |
| Ileum (excluding last 4 in.) | 1 in a newborn child<br>1 at three months<br>8 *at these years*: 3, 5, 7, 8, 8, 11, 14, *and* 62 |
| Ileum (in last 4 in.) .. | 1 in a newborn child<br>2 in first week of life<br>6 *in the first year of life*<br>2 at 5 and 10 years |
| Cæcum .. .. .. | 1 in a fœtus of six months<br>3 in the first six months of life<br>6 *at these years*: 5, 12, 21, 21, 23, 29. |
| Ascending colon .. .. | 1 at eight months<br>2 at 11 and 38 years |

The following facts emerge from this analysis : (1) Cysts of the ileum (excluding the last 4 in.) are discovered usually between the third and fourteenth years ; (2) Cysts of the last 4 in. of the ileum are discovered usually in the first year of life ; (3) Cysts of the cæcum are discovered usually at two periods of life (*a*) in the first six months, (*b*) between twenty and thirty years.

**Incidence in Relation to Sex.**—In 30 of the cases the sex was recorded and a slight predominance in females was noted : females 18, males 12. This ratio was the same in different parts of the intestine.

## CLINICAL MANIFESTATIONS

The clinical manifestations of enterogenous cysts are predominantly of a mechanical nature, and for the greater part occur as intestinal obstruction, which results from stenosis, intussusception, or volvulus. An analysis of the findings at operation discloses the clinical picture which is to be expected.

*Jejunum*: 1 obstruction; 1 volvulus; 1 torsion of the cyst; 1 the cyst was partially torn from the bowel and general peritonitis ensued
*Ileum* (excluding the last 4 in.): 4 obstruction; 2 volvulus
*Ileum* (in last 4 in.): 5 intestinal obstruction; 1 intussusception
*Ileocæcal angle*: 2 intestinal obstruction
*Ileocæcal valve*: 2 intussusception (irreducible)
*Cæcum*: 2 intestinal obstruction; 2 intussusception; 2 thought to be 'acute appendicitis'
*Ascending colon*: 1 intussusception
*Transverse colon*: 1 attacks of acute pain

Thus it is seen that, in 34 cases in which the symptoms were described, there was: intestinal obstruction in 14, intussusception in 6, and volvulus in 3.

## TREATMENT

The operative treatment of cysts in the wall of the bowel is not simple, for in many instances the cyst is discovered after a search for the cause of intestinal obstruction or when an intussusception is reduced and a mass remains at the site; in the latter instance the impression is gained that the reduction is incomplete. In any case the patient is very ill and cannot tolerate any but the simplest procedures and unfortunately these often fail.

The following operations are discussed briefly: (1) Enucleation of the cyst; (2) Excision of the bowel containing the cyst; (3) Marsupialization; (4) Evacuation.

**Enucleation of the Cyst.**—This is the procedure to be adopted in cysts isolated in the mesentery, and these cases present no difficulty. Enucleation is much less successful with cysts in the bowel wall, often failing on account of a firm attachment of that portion of the cyst toward the lumen of the intestine. In some cases the cyst is removed with ease. A perusal of the cases in which enucleation was performed or attempted suggests that success was attained when the cyst was subperitoneal or intermuscular and that failure often followed attempts to enucleate a submucous cyst. It has been mentioned that an intimate attachment frequently exists between the intestinal mucous membrane and the wall of the cyst. This probably explains the failure of enucleation, and it is of interest to note that Edwards removed a cyst from the wall of the cæcum together with some of the intestinal mucosa. An extension of this procedure offers an alternative to the severe operations performed in those cases in which attempts to enucleate the cyst fail.

In collected cases enucleation was attempted in 9, and in these was successful in 5 and failed in 4. Miller, in 1913, recorded 10 cases of enucleation without a death.

**Resection of Bowel.**—In many instances resection of the intestine containing the cyst was performed as a measure of desperation when attempted enucleation failed, and the severity of this operation in a young child was responsible for the high mortality. In this series resection was performed in 14 instances, and in only 3 cases is it stated that the patient recovered. Miller estimated that resection had a mortality of 60 per cent.

**Marsupialization of the Cyst.**—In two instances the cyst was opened, anchored to the abdominal wall, and drained. Both patients recovered, one after a second operation to excise the fæcal fistula which formed through the 'pouch'.

Higgins and Lloyd concluded that "enucleation was the operation of choice. Marsupialization and drainage should be done in the remaining cases."

**Evacuation of the Cyst.**—In one instance only was this performed, and death followed. The danger seems to be the spread of infection from the intestine into the traumatized cyst.

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# THE ORIGINS AND EVOLUTION OF COLOSTOMY

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Wounds of the bowels have been recognized from remote antiquity. Perhaps the earliest reference is to be found in the Book of Judges, where we read how Ehud, a left-handed man, stabbed Eglon the King of Moab—" and his dirt ran out and he died ". Hippocrates regarded all such wounds as ' deadly ', and even Celsus advised that their cure be left to Nature.

It could not, however, have escaped the eye of some more acute observers that sometimes these wounded patients survived, Nature establishing a fæcal fistula. Others noticed that Nature herself in some of her grosser malformations established preternatural openings for the intestinal contents, such as umbilical anus, or vulval or vesical openings for the fæcal discharge. Others again noticed and recorded Nature's surgical methods of cure in strangulated herniæ, when she established by sloughing and inflammation an artificial anal site. The anatomy of such openings was well known, and the afferent and efferent loop and the spur were described by many ancient writers. It is not difficult to believe that now and then some bold spirit dreamed of emulating so great a teacher and of establishing an artificial anus in those cases where obstruction was threatening the life of the patient. Indeed, to stab the colon of a sheep or horse suffering from obstruction is a veterinary operation whose origin is lost in remote antiquity.

We read in Coelius Aurelianus that Praxagore, who lived four hundred years before Christ, after uselessly employing various means for the cure of ileus, made an opening in the belly and incised the bowel to evacuate the contents, and then closed the wound so made. Professor Fine, writing from Geneva in 1797, tells us that in " ancient times " it was the custom to open the stomach to remove knives that had been accidentally swallowed. The same writer speaks in praise of incising a strangulated hernia to give issue to the fæces.

The first deliberately recorded suggestion for enterostomy, however, is that of Littré reported by Fontanelle, the historian to the Académie Royale de Sciences. This erudite compiler, writing in 1710 in the reign of Louis XIV, states as follows (extract reduced) :—

> M. Littré saw in the dead body of an infant of six days a maldevelopment of the rectum. The rectum was divided into two portions both closed and connected by only a few threads of tissue of about an inch long. The upper portion of the closed bowel was filled with meconium. The lower portion was entirely empty. " M. Littré, wishing to render his observations useful, imagined and proposed a very delicate operation in the case where one would recognize a similar conformation. It would be necessary to make an incision in the belly, open the two ends of the closed bowel, and stitch them together, or at least to bring the upper part of the bowel to the surface of the belly wall, where it would never close, but perform the function of an anus. Upon this slight suggestion a clever surgeon could imagine for himself details which we suppress. It often suffices to know in general that a thing may be possible and not to despair of it at first sight."

This idea, so magnificently dreamed and so simply stated, was doomed to lie fallow for sixty-six years. We then read how Pillore, a surgeon at Rouen, performed

cæcostomy for a cancer of the rectum. The account which here follows was found in the papers of M. Pillore, sen., by his son, who looked for them upon the earnest inquiry of M. Amussat, who knew by hearsay of the operation. Considering the time of its compilation and the place of his practice—remote from seats of learning—it is a masterpiece of clinical writing and surgical judgement and daring in the practice of a country surgeon. What follows is a literal translation of the elder Pillore's memoir. Let us here rescue his name and deed from surgical oblivion.

M. Morel, a wine merchant and posting master of Vert-Gallant in the district of Brai, was in the course of the year 1776 taken with difficulty in going to stool. He at first experienced some slight pain in the anal region. These pains became a little greater, without, however, becoming insupportable; but the difficulty with his motions increased to such a degree that he became anxious and determined to come to Rouen for consultation and the necessary remedies. He presented himself to M. Delaroche, a capable physician, who ordered him laxatives and gentle purgatives. These softened the bowel contents and relieved him for some time. But finally, as his difficulties increased daily, he was advised to make use of mercury (or quicksilver) in sufficiently large doses, that by their mass they would overcome the obstacle in the bowel. The patient indeed took two pounds of quicksilver. It was watched for every day but did not appear. The motions became totally suppressed and the belly increased in size from day to day, without, however, being tender or inflamed. In this state of affairs I was consulted. (It was now a month since the patient had taken the mercury without having passed a single drop of it.)

I first examined the rectum, thinking indeed it was there the obstruction would be found, believing it was possibly formed by hardened and incarcerated fæces, as I had often seen to happen; but instead of this species of obstruction I found the upper part of the bowel fixed and scirrhous, forming a very large tumour which totally obstructed the rectum. I tried to pass sounds and cannulæ of all shapes and sizes, continuing my efforts for several days, but uselessly. In this state—that is to say, the patient having passed nothing from the bowel for over a month, and his belly enlarging daily in spite of his most austere diet—I proposed to him that I should make him an artificial anus. He agreed with me and cited the case of a man in his village who for several years had had an artificial anus which Nature had provided following a strangulated hernia. I knew of this case, also of another in a woman and from the same cause.

I was then indeed determined to perform the operation, but as the case was a very delicate one I first asked five or six of my colleagues to see the patient in consultation with me. No one was of my opinion and no one agreed with me. But the patient, a man of great sense, being present at our consultation, prayed my colleagues to show him any other means by which he might be saved. They answered that they knew of none. "Very well," he replied, "it is indeed imperative to operate since my illness is mortal and you know of no other means to save me."

Encouraged by so strong an argument I performed the operation in the presence of my confrères, and of six pensioned pupils who were with me at the time. I chose the cæcum as that part of the bowel most suited to our need, as much by its situation as because it would furnish a reservoir, and by its continual and involuntary action would hasten the evacuation of the intestinal contents. A small plate furnished with a sponge in the shape of a large button and held by an elastic bandage was devised in place of a sphincter, so that the patient could at all times voluntarily remove it when he felt the need, and, by means of a small clyster, he could from time to time cleanse out the reservoir. My patient and I conferred together and thought of all these things before the operation. I then operated.

I commenced with a transverse incision a little above the groin which I deepened above and below to the depth of the cellular tissue. I arrived at the aponeurosis of the external oblique which I incised to the same extent a little above the Fallopian ligament (Poupart's) in order to have at least a good inch of space from the integuments to the cæcum. I made a transverse opening in the muscles and peritoneum almost to the same extent. The base of the cæcum, easy to recognize by its appendix, presented itself—I did not have to search for it. I drew the cæcum out as far as possible and without effort; there held by an assistant and myself, I opened it transversely and stitched it to the two lips of the wound by means of a thread on two needles which I passed from one side to the other. I passed them from

within outwards and pulled out the thread in the middle, thus obtaining two ligatures which I tied above and below over two compresses to press together the edges of the wound. The contents of the bowel came out in abundance. For a dressing I applied burnt charcoal and towels. I used no pressure in order that the issue of fæcal matter might not be interrupted. In fact they ran out in abundance for several days, and the belly diminished considerably in size. As the quicksilver was giving us anxiety and we had not seen a single drop of it appear, we caused the patient to be put in all possible positions that might give it an easy issue. There was not the slightest sign of it, however. Fourteen or fifteen days had passed since the operation, during which time the wound had suppurated and the bowel was glued to the skin. I had taken out the stitches and all appeared to be in the best possible state when the patient reported vague pains in different parts of the belly. We at first attributed it to gases shut in the intestines, but the patient, uneasy, said always that the pains were due to the mercury and consequently continued to take such positions as might help it to come out. On the twentieth day the belly, which had been very flat, became swollen and painful. Emollient fomentations were applied, and through our artificial anus we threw some injections into the colon. He was bled twice, but in spite of all our efforts the symptoms quickly augmented and the patient died on the twenty-eighth day after his operation.

I performed the autopsy in the presence of the same surgeons, colleagues, and pupils, and found as follows :—

The cæcum and the whole of the colon were healthy and in good condition. The cæcum was adherent to the lips of the wounds, except in one angle where there was a small area of suppuration in the neighbouring cellular tissue, which did not, however, communicate within. The colon was open to the whole of its extent, and only contained some glairy mucus. The cancerous obstruction which was the primary illness was eight or nine inches long, situated at the end of the colon and the beginning of the rectum, totally obliterating the intestinal canal. The tissues surrounding the rectum were hard and fixed. At the side of the rectum was an opening whose calloused edges announced it to be a species of chancre from which issued fæcal and purulent material. The peritoneum in the neighbourhood of the kidney was inflamed, without, however, having suppurated. The peritoneum was inflamed and adherent to the folds of the intestines. The quicksilver which the patient had taken was found in one of the last convolutions of the jejunum, which it had dragged down by its weight to the pelvis, behind the bladder. It was pocketed in that portion of the bowel which contained it. This bowel presented here and there gangrenous areas, and was inflamed, the inflammation extending to the loins. The mercury was all recovered and it had not lost a bit of its weight. We believe that we could conclude that if the operation has not met our expectations for its success, it was because of the mercury. For it is very probable that when the intestines, which because of their great dilatation had lost their power of action, became empty of stercoral material, their peristaltic action was not sufficiently powerful to move the mercury. Then followed inverted and retrograde movements, as announced by the nausea and colic which the patient experienced on the twentieth day of his illness. Considering the pull on the mesentery and intestines by this mass of two pounds, one is not surprised that gangrenous inflammation occurred and produced the death of the patient.

Surely a remarkable case and one which any modern surgeon would not be ashamed to report.

Allan, the recorder to the Recueil Périodique de la Société de Médecine de Paris, writing in 1783, tells us that by *hearsay* he learnt that Dubois performed Littré's operation for an imperforate anus in a child of three days. The child died on the tenth day.

Colostomy, however, had its real birth when Duret in 1793 performed successfully left iliac colostomy for a case of imperforate anus in a child three days old. Here is his account :—

Friday, Oct. 18, 1793, Marie Poulaouen, midwife of Brales, delivered the wife of Michel Ledréves, labourer, of a child. She noticed the infant had no anus and that the sexual parts were malformed; judging that in this state of affairs the child had not long to live she advised the parents to bring the child to Brest to receive surgical aid. On Saturday at

ten in the morning the father came to my house and I examined the child. The sexual organs were so formed that the scrotum was divided at the raphé into two equal parts, each containing a testicle. At first sight one believed the child to be a female. The glans penis lay upon the perineum pierced by the urinary meatus from which urine issued freely. The region of the anus showed no sign of the existence of a rectum. The skin was of natural consistency and colour, and no tumour presented when the child strained. After making this examination I believed the case to merit the attention of those most skilled in the art of healing, and with this in view I called a consultation of all the physicians and surgeons attached to the various hospitals of the city. The consultants advised opening the skin at the spot where the rectum should be present and searching for the bowel. The operation was not successful. I was able to appreciate by passing a sound through the wound into the pelvis that the lower portion of the big bowel was absolutely missing. It was now four in the afternoon and the infant appeared without resource. The vomiting, the extraordinary swelling of the belly, the coldness of the lower limbs seemed signs of certain death. To my surprise, however, the next morning the child still lived. This decided me to call a second consultation, at which I proposed as a last resort, to prolong the life of the child, the performance of laparotomy and the establishing of an artificial anus. To give me confidence in this most extraordinary procedure I performed it upon the dead body of a child of fifteen days which I took from the poorhouse of the city. I made an incision on the left side between the last of the false ribs and the iliac crest about two inches long. I exposed the pole of the kidney and a portion of the left side of the colon; this last was opened. I then injected some water by the anus. A portion of the fluid came out through the opening of the colon and a portion escaped into the belly. I then recognized by opening the belly that in the fœtus the lateral areas of the colon are not extraperitoneal as in the adult, but that the colon has a mesocolon which renders it free and floating. This circumstance caused me to reject the operation in this region in the fear that it would give rise to an escape of meconium into the belly. Those assembled after witnessing this trial and after prolonging the discussion sufficiently to prove both its interest to humanity and to surgery, decided: (1) That without some extraordinary intervention the death of the child was inevitable; (2) That the axiom of Celsus, "that it is better to employ a doubtful remedy than to condemn the patient in certain death", here found its application; and finally (3) That the decisions of M. Hevin upon laparotomy were not transgressed by this operation, as the cause and course of the malady were, as here, recognized.

I opened the belly of the little patient in the left iliac region in the neighbourhood where the sigmoid colon was forming a tumour a little apparent to the eye and where the meconium already imparted a slightly deeper colour to the skin. I made an opening about an inch and a half long which served for me to introduce the index finger into the belly, with which I lifted and pulled out the sigmoid colon. In the fear that it would immediately fall back into the belly I stitched it by two wax threads passed through the mesocolon. I then opened the colon longitudinally. Gas and meconium came out in abundance. When the bowel had emptied itself to a certain extent I applied a dressing. It was simple and was composed of a pierced compress. In the night between Sunday and Monday the baby slept well, the body heat returned, the vomiting ceased, and the child took the breast easily on several occasions. The day following the operation all who had witnessed the operation the evening before expressed themselves satisfied with the advantageous changes they perceived. The bandages which surrounded the child were filled with meconium, and his voice, which had previously hardly been distinguishable, was now heard lustily.

On the third day, as things were going from better to better, I charged the parents to bring the child twice daily to the hospital. Citizen Massac, Chief of the Administration, and Citizen Coulon, Physician in Chief, were charged to provide the necessary dressings.

On the fourth day the stools became yellow and less in quantity, so I ordered a washing out with simple water and two drops of syrup of rhubarb. This produced a good effect and gave the patient several stools.

On the fifth day the threads which held the bowel appeared useless, so I removed them, for they were already producing redness and irritation in the region of the artificial anus.

On the sixth day about an inch of the internal coats of the bowel appeared through the opening, giving the wound the appearance of a chicken's egg. I attempted to reduce the prolapse by passing a lead cannula into the fistula, both to obstruct any further herniation and keep a free passage for the fæces, but the child's cries made one defer this means. The

instrument has, however, since then been perfected by Citizen Morier, a clever cutler of this city. On the seventh day the child was so well, both at the site of operation and in exercise of his functions, that I judged him no longer in need of care or supervision by a person of art.

It is interesting to note that this little patient lived to the age of forty-five.

With Duret begins the history of colostomy. Observe: (1) Duret antedated Callisen in the conception of lumbar colostomy; (2) Duret was ignorant of Littré's academic suggestion of left iliac colostomy; (3) He made a very small incision; (4) He made the bowel secure by a stitch in the mesocolon, thus obviating recession of the bowel—in this idea he anticipated the proceeding of Allingham, jun., by over ninety years; (5) He noted the occurrence of prolapse; (6) He used the artificial anus as a channel for the administration of a colonic wash-out. Surely, as the prophet says, " All knowledge is but a remembrance, and all discovery but a forgetting ".

For all that Duret was a Professor of Surgery at the Military and Marine Hospital at Brest, he was but a humble and obscure naval surgeon practising in a remote seaport, and his experiments, his operation, and his remarks were lost to surgical knowledge until rescued by Amussat some forty years later. He had the bitterness to know that his thunder was stolen by a contemporary, one Dumas, Professor of Medicine at l'Ecole de Santé de Montpellier.

Dumas in 1797 reported to the Medical Society of Paris a case of imperforate anus. The surgeon Estor, he stated, made repeated attempts to find the rectum from the perineum but failed. Dumas then suggested to Estor the practicability of establishing an artificial anus in the left iliac region. Dumas states that Estor replied, " I approve of the idea, but I have not the courage to execute it. The parents are against it and I shall compromise my reputation by practising so severe an operation of which the success seems so uncertain." The child died on the third day and Dumas made an autopsy. He made an incision in the left iliac region, the colon was distended with gas and liquid and occupied a considerable space. It was pressing hard against the belly wall. Dumas states it would have been very easy to make the artificial anus which would have prolonged the patient's life and " which would have been a rare and interesting contribution to science ". States Dumas, " Past historians have contented themselves with describing the malformations of the anus and the rectum, but they give no indication for a cure ". He pleaded that the method of Nature as seen in preternatural anus and in the artificial anus following strangulated hernia is an indication of the method surgical art should employ. He stated that the " iliac passion " following upon obstruction of the rectum could be relieved by this means. The operation he suggested was an opening of the belly in the region of the lower end of the colon, then to open the intestine and stitch the opening by means of a thread. The incision ought to follow the direction of the muscle fibres, which would preserve a sphincteric action and avoid either collapse or prolapse of the bowel.

Dumas held a high position. He was a professor of medicine in a big school. That he was by no means ignorant of the literature of his time is conclusively proved by the two pages of quotations and references upon rectal malformations which he gives in his essay. It is incredible for us to believe that he had not read of the proposals of Littré, and still more incredible that he was ignorant of the report of Duret's case which was published in full in a medical periodical which was distributed to the whole of Europe. Indeed, were he ignorant of both these

proceedings it is hard to believe that he had not heard of the operation already performed by Desault, Surgeon-in-Chief to l'Hôtel Dieu in Paris, who in 1794 performed Littré's operation in a case of imperforate anus two days old. It is interesting to note that Desault did not stitch his wound. He operated through an incision $3\frac{1}{2}$ in. in length. After opening the bowel he stuffed in a dressing with the object of " keeping open the passage for the meconium and causing the edges of the wound to stick ". The child died in four days. The specimen was modelled in wax and is in Desault's Museum.

No. Dumas stole the thunder of Duret and maintained a discreet silence regarding Duret's operation, and appeared before the Medical Society of Paris in borrowed plumes.

This case of Dumas', however, upon which he did not operate, created great attention in France and Europe generally. It was the subject of a review by Allan, the Recorder of the Medical Society of Paris, and by Martin, the Recorder of the Medical Society of Lyons, who were more generous in their appreciations of the surgeons who had actually dared to operate. It was the consensus of medical opinion that the inguinal incision of Littré was infinitely preferable to the vertical lumbar incision of Duret and Callisen. They believed that it was an impossibility to approach the colon posteriorly without opening the peritoneal cavity. Allan alone of all commentators does not despair at this time of the lumbar route, and states in 1797 " that the lumbar route merits consideration, and, if it could be rendered safe and easy, the results would be infinitely less disagreeable than those of the abdominal or iliac route ".

The next step forward in the history of colostomy leads us to Geneva. In 1797 Professor Fine, Surgeon-in-Chief to the Hospital of Geneva, performed transverse colostomy in a woman aged 63 years for acute obstruction due to a cancer of the rectum. Fine stated that for many years he had been struck by the insufficiency of medical means in tympanites and had long been determined to perform enterostomy. He had often advised it but had never been allowed to perform it. In the case reported he had intended to open the ileum, as he states it is here that Nature most often makes an artificial anus in strangulated hernia. His patient being fourteen days obstructed, he opened her in the middle line below the umbilicus, drew out a loop of inflamed bowel, passed a stitch through its mesentery, and sewed it to the skin. His operation was successful. The patient died three months later, when he discovered at the autopsy that he had performed a transverse colostomy. The patient had a growth in the upper part of the rectum causing complete obstruction. In his report he stated that he was ignorant of Dumas' paper and only received his copy of the medical periodical two weeks after having performed this operation. Fine's two memoirs are models of correctness. Professor Fine was conversant with all the history of this time and acknowledges all previous operators and writers. He gives generous tribute to Dumas for his idea of enterostomy for cancerous obstruction.

We now leave France in our pursuit of colostomy and turn to Copenhagen. Here we find that Callisen, Professor of Surgery at Copenhagen, in writing upon imperforate anus states ; " If the intestines (rectum) cannot be reached either with the knife or a trocar, it will indeed be difficult to save the patient. An incision of the cæcum or the descending colon has been proposed in these cases, by means of an incision in the left lumbar region at the border of the quadratus lumborum

to establish an artificial anus; the chance of success is very slight and the life of the little patient can hardly be saved. However, the intestine ought to be more easily reached in this spot than from the inguinal region." This observation is to be found in Callisen's *Systema Chirurgæ Hodiernæ* (p. 688) published in 1800. Sabatier, writing in 1810, states: "Instead of making an opening in the belly above the groin, M. Callisen, a surgeon who enjoys a distinguished reputation at Copenhagen, has proposed to search for the descending colon in the left lumbar region, where he supposes that it is to some degree outside the general peritoneum cavity. Callisen attempted this operation on the dead body of an infant with an imperforate anus. He did not carefully observe his landmarks, with the result that he opened the peritoneal cavity and penetrated the abdomen. Having made a second incision more posteriorly he came upon the colon, as he had hoped. However, he does not disguise the fact that his fingers, which he introduced into the first incision to help him secure the bowel, were very useful in performing the posterior operation. The wound which he made does not appear to him to be of great importance in view of the small number of blood-vessels which he encountered."

We note that Callisen does not claim the idea as being original. Yet it is he who has had the credit of the conception of lumbar colostomy until now. It was, however, Duret who first originated this incision in 1793. Callisen thought very little of the lumbar method of approach, as indeed did all who followed him until the time of Amussat in 1839.

The first colostomy in England was practised in 1815 by Freer, a surgeon in Birmingham. Freer was also the first surgeon to tie the external iliac artery successfully. In 1815 he performed left iliac colostomy for imperforate anus. The child lived three weeks, dying eventually of marasmus. In 1817 he performed left iliac colostomy in a man of 45 suffering from complete obstruction due to what appears to have been a simple stricture of the rectum. This patient died on the ninth day. His colostomy had acted freely for the whole time. The patient had been purged every day and bled once. From a perusal of the notes of this case he seems to have died of a rupture of the cæcum induced by excessive purgation. This accident is not unknown at the present day. Freer's incision was an oblique one in the left iliac fossa. The colon was stitched to the wound, the stitches were removed on the fifth day, as by then the gut was adherent to the wound. On the eighth day the bowel prolapsed. Several enemas were given through the artificial anus. Freer states that the opening in the belly was made very small to prevent extrusion of the bowels, as this was an accident which had frequently happened to him when operating on dogs.

The next operation in England was performed by Daniel Pring, a surgeon of Bath, who in 1820 performed left iliac colostomy for a patient who was twelve days obstructed from cancer of the rectum. He was led to perform the operation in imitation of the artificial anus which follows the natural cure of strangulated hernia. He was ignorant at the time of any previous writing upon the subject. His patient lived. Pring concludes his memoir as follows: (1) The operation prolongs life in cancer of the rectum; (2) It is always applicable in imperforate anus; (3) It would be useful in simple strictures of the bowel; (4) Peritonitis is a danger—but this would not deter him from operating; (5) It is imperative always to open distended bowel; (6) He advises cæcostomy for obstruction in the transverse colon; (7) The opening can be controlled to act once in twenty-four hours; (8)

Prolapse can be prevented and the opening protected by means of a truss and pad. Pring seems to have been a clever, thoughtful, and resourceful surgeon.

No further operations were performed in England until 1824, when Richard Martland, Physician to Blackburn General Dispensary, operated successfully for a chronic intussusception. His operation was quite successful.

It was not, however, until the time of Amussat that colostomy as an operation was rescued from the realm of the occasional and heroic and advanced to its proper place in surgery. Amussat was a clever and well-known surgeon in Paris. He was called to attend the celebrated Professor Broussais in his last illness. He stated that on seeing him die from stercoral tympanites produced by a rectal carcinoma, he determined that never again would he remain a passive spectator of such another death. The death of Broussais, and the examination of two specimens in the Musée Dupuytren—the one a fungating rectal growth, and the other a scirrhus of the rectum removed post mortem from the body of the tragedian Talma—led him to a careful reconsideration of the whole question of artificial anus. He collected the statistics of all cases from Pillore in 1776 to his own first case in 1839. Over a period of sixty-three years there were 29 cases, with a mortality of 20. Of these 29 cases, 21 had been operations for imperforate anus, of which only 4 survived. It is curious to note that all these 4 survivors had been operated upon in Brest, the seaport town in which Duret first performed the operation. The remaining 8 cases were adults, and of these only 5 had survived. All of these 29 cases had been operated upon by the abdominal route. Amussat attributed the death in all cases to peritonitis.

Amussat, in spite of the failures of Duret and Callisen, and the condemnation of all previous writers and the surgeons of his own time, determined to decide for himself the possibility of opening the colon posteriorly without wounding the peritoneum. By a series of dissections he showed conclusively that Duret and Callisen, while recognizing the peritoneal relationships of the colon in the lumbar region, failed to appreciate the possibilities of lumbar colostomy in the adult. He demonstrated the existence of large cellular spaces in the lumbar regions and in the pelvic mesocolon, and that these spaces are greatly developed both in children and adults under conditions of obstruction, or of deliberate distension by means of a bellows and tube. He showed that both in the infant and adult lumbar colostomy was at all times possible.

Amussat abandoned the longitudinal incision of Duret and Callisen. He operated by a transverse incision 4 in. long midway between the last rib and the iliac crest, and extending well back to the erector spinæ and the quadratus lumborum muscles. The bowel was fixed at the anterior angle of the wound by a stitch and the rest of the wound left open. He operated without assistance, and twisted the arteries instead of tying them, as the long ligatures which were then used got in his way. His guide to the colon was the pad of fat at the outer edge of the quadratus, and his guide to the point to open the bowel was the 'yellow spot' where the peritoneum is reflected to the abdominal parietes. If obstruction was not already present, Amussat distended the bowel by means of a bellows and tube. He contended that the operation was as urgently indicated in cases of obstruction of the big bowel as was herniotomy in strangulation. He determined the site of obstruction by: (1) Rectal examination; (2) Estimation of the amount of fluid it was possible to inject in the rectum; and (3) In some cases

when the exact site of the tumour could not be determined he punctured the distended bowel with a small trocar.

Amussat advised operation on the right side: (1) When the tumour was too near the site of operation on the left side; (2) When the obstruction was far from the anus; and (3) When the site of obstruction could not be determined.

He states that in right-sided lumbar colostomy the retrograde peristalsis of the colon distal to the opening is a source of distress, and that " it would be advantageous to form some sort of septum to prevent the fæces passing beyond the opening ". This, however, he could not do, as he states the intestine is so strongly fixed to the edges of the wound that to form a spur would be impracticable and dangerous. He states also that there is no tendency to prolapse in his method, and that the smallness of the opening, which has always a tendency to close and retract, prevents the continuous and involuntary escape of fæces. He performed the operation nine times successfully.

Lumbar colostomy was an operation well suited to the pre-anæsthetic and pre-antiseptic times in which Amussat lived. His activities gave the operation an impetus which carried it successfully to the time of the younger Allingham. It became until Allingham's time the operation of election, and was practised by all surgeons in Europe, England, and America.

No proctologist should be in ignorance of Amussat's writings. His memoirs deserve to be read by all surgeons. They are models of all that surgical communications should be in honesty, completeness, conciseness, and well-reasoned judgement.

Amussat concludes " An artificial anus, it is true, is a grave infirmity, but it is not insupportable. To be able to practise it a surgeon ought to fear to be surprised by a pressing occasion, and he should prepare himself by many repetitions of the operation upon the cadaver."

The first operation of lumbar colostomy in England was performed by Clement, of Shrewsbury, who in 1841 did right lumbar colostomy for a whipcord stricture of the transverse colon. The patient lived three years. Lumbar colostomy, however, owes its real introduction in England to John Erichsen, Surgeon to University College Hospital. Erichsen was a pupil of Amussat and witnessed Amussat's first operation. Hence his prejudice in its favour. In a report published in 1841 he gave Amussat's writings to the English medical public. He advised the operation for: (1) Imperforate anus; (2) Simple retention of fæces which could not otherwise be relieved; (3) Obstruction of the big bowel; and (4) In cancer of the rectum as soon as pain becomes severe. He claims for the operation that the wound is easy to dress, that its posterior situation is less distressing than is the inguinal, and that the absence of the spur is indeed an advantage, as the wound may close naturally.

A further extension of the applicability of lumbar colostomy is seen when Pennell in 1849 performed it for rectovesical fistula, following a rectal stricture and a fistula in ano. His patient lived for fifteen years. Croker Pennell was a Lecturer in Surgery at Westminster Hospital and later was attached to the Faculty of Medicine in Rio de Janeiro. In 1850 Avery as Surgeon of Charing Cross Hospital performed left lumbar colostomy for a case of intestinal obstruction of nine days' duration. The patient died the next day, and the autopsy showed a volvulus of the cæcum and descending colon. This shows the limitation of the operation apart from certain diagnosis as to nature and seat of obstruction. Luke,

a Surgeon to the London Hospital, appreciated this fact, and writing in 1850 he stated that lumbar operation was only useful when the seat of the obstruction was definitely recognized. Luke was the first surgeon to perform a pararectus incision and to bring out the bowel in the neighbourhood of the rectus muscle. His colleague, John Adams, of the London Hospital, also operated by a hypogastric incision and brought the bowel through the linea alba. Adams advised operation in all cases of cancer of the rectum before obstruction.

Curling, Consulting Surgeon to the London Hospital and President of the Royal College of Surgeons, expressed himself in 1851 as a strong partisan of the abdominal operation. He advised colostomy for stricture of the rectum and for cancer of the rectum before obstruction. He stated that excision of the rectum for cancer had no place in surgery. He regarded both the lumbar and abdominal operations as equally dangerous, but preferred the abdominal route as being easier, as giving a spur, easier to dress and control, and states that by the abdominal route the bowel can always be found. In 1865, however, he appears to have recanted and he became an ardent advocate of the lumbar operation. He apologetically states that with the lumbar operation there may be an occasional sphincteric action by the lumbar muscles. This communication of Curling's in 1865 gave a tremendous impetus to lumbar colostomy in England, as did also the writings of Cæsar Hawkins in 1852.

The next advance in colostomy was when Nathaniel Ward in 1865 performed lumbar colostomy for rectal cancer as a routine upon recognition of its presence.

There were two staunch advocates in England of lumbar colostomy in the persons of Allingham, sen., and of Thomas Bryant. William Allingham, writing in *St. Thomas's Hospital Reports* in 1870, and in his book, *Diseases of the Rectum*, in 1873, advocated lumbar colostomy for insurmountable obstruction or intense pain due to a cancerous growth, and for rectovesical fistula in the male. In all his writings he showed great ignorance of the history of the operation, and denied indeed that Amussat ever performed it.

Thomas Bryant, Surgeon to Guy's Hospital (and a great advocate of the galvano-cautery knife), writing in 1874, stated of lumbar colostomy that it prolonged life, eased pain, and prevented aggravation of the disease in cancer of the rectum. He also advised it as the best possible means of treatment in incurable fistula and abscesses of the rectum.

Bryant to the end of his days remained a loyal exponent of the lumbar operation. Speaking in 1889 in his Bradshaw Lecture, delivered at a time when the writings of Allingham, jun., Reeves, and Cripps had revived for all time the operation of inguinal colostomy, he stated that the abdominal operation was not proved superior to the lumbar operation. His lecture, indeed, is a thinly veiled attack delivered by an old conservative surgeon against younger and more active men who were carrying the banner of surgery one step forward. Bryant operated by an oblique incision. He claimed for his operation that it was safe and gave a good spur. He operated to relieve symptoms and did not wait for obstruction to appear.

The 1880's witnessed an increasing interest in the abdominal operation. Peritonitis was ceasing to be the surgeon's bane. Abdominal surgery was making rapid progress, and it is but natural that surgeons gave an added attention to the possibilities of colostomy by the abdominal route. The operation was begging to be better understood, and many surgeons have given their names to proceedings

which testify to their ingenuity in devising methods of spur formation, the prevention of prolapse, and the establishment, with varying success, of some form of sphincteric control. It will be remembered that Duret stitched the mesocolon. This most effectually prevented collapse of the bowel and formed a good spur. Although in 1833 Dupuytren, reviewing the work of Petit, Scarpa, and others, established for all time the characteristics of an artificial anus with its afferent and efferent loops and its spur, and distinguished artificial anus from fæcal fistula, it was not until the time of Allingham and Bryant that a definite effort was made to establish any sort of spur, and this only in lumbar colostomy, and with indifferent success. Jones in 1866 suggested formation of a spur in lumbar colostomy by dissecting up the mucous membrane and stitching it to form a bridge between the two loops. Later on Madelung suggested complete division of the bowel with closure of the lower loop, which he allowed to fall back into the wound. In 1841 Schitzinger applied the principle of closure and invagination of the lower loop to the abdominal colostomies. It was not until 1855 that this primal necessity of a spur was definitely recognized, when Knie, a surgeon in Moscow, experimenting on dogs, found that he could make a slit in the mesocolon, through which he was able to join all the layers of the belly wall. This idea he used successfully in operation upon the human being. In 1887 Ball, of Dublin, in describing his operation for colostomy stated that he always stitched the mesocolon to the skin. This, Ball states, he did to prevent recession of the bowel, not, however, with the view to the formation of a spur. In 1887 Allingham, jun., definitely described the mesocolic stitch. His stitch was a half mattress securing the mesocolon to the skin. He describes it as an essential part of his operation. It is curious that Cripps, writing at the same time, did not appreciate or use this mesocolic stitch. In 1888 Maydl advanced and simplified colostomy with his idea of passing a vulcanite rod through the mesocolon. This rod lay upon the belly wall and very effectually prevented recession of the bowel. Kelsey, a well-known New York surgeon, in 1889 described independently a method of securing a spur and preventing recession of the bowel. He passed a hare-lip pin through all layers of the abdominal wall on one side, then through the mesocolon, and then through the abdominal wall on the other side. Reeves, a Surgeon to the London Hospital, described a somewhat similar method in 1892. Paul, writing in 1890, said that he considered lumbar colostomy " prompt, safe and suitable " to relieve acute obstruction. He considered the inguinal route easier, but, in acute cases, believed it to be a great source of danger because of the unavoidable risk of fæcal infection. He then described his tube, which he tied in the proximal end of the bowel.

In Littré's original suggestion in 1710 he advocated in imperforate anus that the closed proximal end of the bowel should be brought to the skin, " to make for ever " an artificial anus. In the endeavour to perfect colostomy, to obviate the spur, and prevent prolapse, surgeons began to practise complete division of the bowel with closure of the distal end, which was allowed to fall back into the abdomen, whilst the proximal end was secured to the belly wall. This proceeding, first practised by Schitzinger in 1881, then by Senn, of America, was in 1889 adopted by Jessop and Purcell; by Paul, Braun, and Weir in 1890, and with various modifications by Ryall and Lilienthal; and in 1909 by Marro, of Turin.

Allingham, jun., Cripps, and Reeves re-established inguinal colotomy, not forgetting the writings of Ball, of Dublin, and of Davies-Colley, Surgeon to Guy's

Hospital. Davies-Colley made two valuable and original suggestions. The first was that of delayed opening of the bowel—the operation ' à deux temps '. The second suggestion he made was that of exteriorization of the colon when it is the seat of a malignant growth. Davies-Colley deserves well to be remembered for so ingenious a suggestion. It was enthusiastically adopted by Verneuil, of Paris, and is described in full in the writing of Réclus. Verneuil, by the way, was accustomed to stitch together for the extent of some inches both loops of the bowel, so that on being opened they presented the appearance of a double-barrelled gun. Ball, of Dublin, writes in 1887 as an advocate of what he terms ' laparo-colostomy '. He believed in an abdominal exploration before colostomy. He stated that it was easier to find the bowel, that the exact spot for the colostomy could be selected, and that the operation was made easier by exploration.

We now recognize that there have evolved two distinct methods of performing an abdominal colostomy. The one depends for its efficiency upon a spur—the method of Duret, Allingham, Knie, Madyl, Kelsey, and Reeves. The other method is that of division of the bowel, closure, and invagination of the distal end, and utilization, in various ways, of the freed proximal loop. It was, by the way, the practice of this latter method which led surgeons to a careful investigation and appreciation of the colonic blood-supply. Many ingenious operations have been devised to obtain sphincteric control of the anus which result from this method. Meanwhile in our consideration of the first category we must not forget to mention the names of Ward, of Leeds, Hartmann, of Paris, and Miles, of London. Ward, who was a colleague of Lord Moynihan of Leeds, devised a suture known as Ward's mattress suture. It is a further step in Allingham's mesocolic suture. Hartmann operates through a muscle-splitting incision such as is employed in McBurney's operation. He forms his spur by passing a roll of gauze through the mesocolon, after the method of Zuckerkandl. And latest of all is the method of Miles, who, first of all, explores the abdomen through a median subumbilical incision and mobilizes the proximal portion of the pelvic colon by dividing any adhesions that may exist. In this way he ensures that the segment of pelvic colon utilized for the colostomy adjoins the descending colon. The loop of colon so mobilized is withdrawn from the abdomen through a small stab incision situated at the junction of the outer and middle thirds of a line drawn from the umbilicus to the left anterior superior spine of the ilium. A loop of stout silk is passed round the mesenteric vessels and then through a loop of silkworm gut inserted through the skin at a point midway between the umbilicus and the stab incision and tied, not so tightly as to occlude the arteries but tightly enough partially to obstruct the venous return. In this way a solid œdema of the spur is produced which effectively prevents it from receding.

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# *EXPERIMENTAL SURGERY*

## THE RELATION OF THE EXTRINSIC NERVES TO THE FUNCTIONAL ACTIVITY OF THE ŒSOPHAGUS*

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THE purpose of this investigation was to determine the exact distribution and function of the extrinsic nerves to the œsophagus and cardiac sphincter, and thus to obtain if possible a rational basis for the surgical treatment of achalasia.

**Action of the Vagus on the Œsophagus.**—It is generally agreed that vagal stimulation causes an increased 'tonus' of the thoracic œsophagus, but different workers have obtained varying effects on the cardiac sphincter after vagal stimulation. Openchowski[1] described a 'dilator nerve' to the sphincter. May[2] and Langley[3] showed that the dilator effect was obtainable by stimulation of the vagus as a whole and was not due to a separate dilator nerve. Koennecke[4] and Rieder[5] obtained the reverse effect. Veach[6] reported that sphincter closure followed low-frequency stimulation, while high-frequency stimulation inhibited the sphincter. Carlson, Boyd, and Pearcey[7] related the variable effects to the coexisting gastric tonus, a point of view which is shared by McCrea, McSwiney, and Stopford.[8]

Reid[9] in 1839 noted that in rabbits vagal section in the neck caused stasis of the food in the œsophagus. It is generally agreed that this stasis is due to increased tonus of the cardiac sphincter followed later by dilatation of the œsophagus (Bernard,[10] Schiff,[11] Kronecker and Meltzer,[12] D'Almeida,[13] and Klee.[14]) Krehl[15] has described a patulous cardia following vagal section in the neck. McCrea, McSwiney, and Stopford[16] considered that the increased sphincter tonus was only temporary, but it must be noted that in all their experiments the vagi were divided after they had passed into the abdomen.

Carlson,[17] quoting Cannon,[18] stated that after bilateral vagotomy in cats Cannon noted obstruction in some cases for several days and in others indefinitely. Cannon[18] distinguished two different effects of vagal section: (1) paralysis of the upper zone which contains striped muscle, and (2) in the lower unstriped zone paralysis followed by recovery of peristalsis in response to the local stimulus of mechanical distension. In relation to obstruction he stated: "In my experience a difficulty in forcing food through the cardiac sphincter explains to some extent the slower emptying of the gullet during the first few days after operation, which is proved by the fact that strong peristaltic waves, so strong as to produce a very marked bulging of the tube in front of them as they advance, have failed to force food into the stomach. In my experience this usually does not persist as a considerable

* From the Buckston Browne Surgical Research Farm.

obstacle and the forcing of food into the stomach by œsophageal peristalsis becomes in time not difficult. But there have been instances under my observation in which there was continued trouble. . . . The œsophagus in these cases suffered a marked dilatation and became filled with food which decomposed." Such a description could be interpreted not as that of a transient obstruction but rather as an obstruction overcome by the recovery of peristalsis.

Jurica,[19] in repeating this work, has described the recovery of irregular movements in the upper zone as well, which he believes are mediated through the ganglia of Auerbach's plexus, and which he has demonstrated in this region.

**Action of Sympathetic Nerves upon the Œsophagus.**—Gaskell[20] has denied that the œsophagus receives any sympathetic supply, and states that all observers agree that the splanchnic nerves do not affect the œsophagus in causing either contraction or relaxation. Carlson,[17] however, obtained both contraction and relaxation of the cardia on splanchnic stimulation, and Veach[21] obtained an occasional motor response. Page-May,[22] however, could obtain no response.

Kuré[23] and his fellow investigators, experimenting on dogs, find that stimulation of the stellate ganglion does not cause contraction of the œsophagus, but that stimulation of this ganglion during vagal stimulation results in an increase of the contraction of the œsophageal wall. Rieder[5] was unable to obtain this result or any sympathetic response from the œsophagus or cardia. His experiments were conducted in rabbits and dogs.

As regards excision of the sympathetic supply, Jurica[19] found no change in the X-ray appearances of the œsophagus following double splachnectomy. Kuré[24] has stated that excision of the stellate ganglia is followed by stoppage of a barium meal in the part of the œsophagus behind the heart, and excision of the sympathetic fibres passing to the cardiac sphincter in an animal in which both vagi had been divided increased the obstruction at this site resulting from the vagal section. On these grounds he postulated synergism of the vagus and sympathetic in the œsophagus. Jurica[19] found that previous double splanchnectomy, followed by bilateral vagal section, does not alter the appearances resulting from the vagal section.

Kuntz[25] states that in man the œsophagus is innervated chiefly from the inferior cervical ganglia, further branches also passing to the œsophagus directly from the thoracic sympathetic ganglia, or from these ganglia via the aortic plexus or a plexus in the posterior mediastinum. It is in relation to these direct branches that one discrepancy arises, some authors figuring many and others few or none. Hovelacque[26] considers that they are few in number and not to be found in all subjects. Where they occur they are more numerous above the origin of the great splanchnics. With the latter point Testut[27] is in agreement.

In regard to the possibility of a recurrent supply from the abdomen, it might well be that those sympathetic fibres which supply the cardia also innervate the adjacent sphincter; and here Brandt[28] has described a large branch arising from the semilunar ganglion of the left side passing directly to the cardia. Hovelacque,[26] in describing the coronary plexus of the stomach, states that it is distributed to the cardia with the cardio-œsophageal branch of the left gastric artery. Hurst[29] observes that branches derived from the splanchnic accompany the inferior phrenic arteries and piercing the diaphragm supply the lower part of the œsophagus.

**Nature of the Cardiac Sphincter.**—This is the subject of controversy. Evidence for the existence of such a structure has been brought forward by Langley,[3]

and more recently by Poulton and Payne,[30] who have observed the contraction occurring at this region in rabbits and have caused relaxation of the contracted region on vagal stimulation. McSwiney[31] regarded this structure as part of the stomach rather than the œsophagus, on account of the response of its musculature to drugs, the responses resembling gastric rather than œsophageal reaction. Views held by other authors suggest that sphincteric action in this region depends upon the contraction of adjacent structures—e.g., the cruciate fibres of the diaphragm and the so-called liver tunnel, either alone or together with an intrinsic sphincter mechanism. Thus Jackson[32] stated: "The diaphragmatic pinchcock is the normal mechanism by which, along with kinking of the œsophagus, the food in the stomach is prevented from regurgitation, and the diaphragmatic pinchcock opens at the proper moment in the deglutitory cycle." Mosher,[33] in describing the action of the surrounding liver in relation to deglutition in the abdominal œsophagus as observed by X rays, writes: "In watching a patient with a normal œsophagus swallow, the œsophagus is seen to come to a point momentarily at the upper border of the liver. Then, after a delay of a second or two, the liver tunnel opens up and the milk streams into the stomach."

Fulde,[34] in a recent investigation of the mechanism of the cardia, describes an intrinsic mechanism the action of which is modified by the phrenico-œsophageal membrane. This structure, in virtue of its high attachment to the œsophagus, can close the cardia by compression during descent of the diaphragm, opening therefore occurring at the beginning of expiration. This action is mechanical and depends upon the relative position of cardia and diaphragm.

## EXPERIMENTAL METHODS

In attempting to elucidate some of these problems the methods chosen have been anatomical dissections in stillborn children and animal experiments. In animals the methods of investigation were two.

**Method 1.**—Firstly, the study of changes produced in the intra-œsophageal pressure on electrical stimulation of the extrinsic nerves to the œsophagus. The changes of pressure produced were received by a thin rubber bag introduced through the mouth and connected by a tube to a pump by which the bag could be distended, and also to a water manometer recording on a kymograph. Electrical stimulation was carried out using a coil and vulcanite insulated electrodes. The coil was kept at 8 to 10 cm., having four volts in the primary circuit. For sympathetic stimulation a combination of an electric pendulum interrupter in the primary circuit and a metronome in the electrode circuit was arranged so as to produce volleys of six make-and-break shocks occurring three times a second. For vagal stimulation it was found that the results were the same if the vagi were left intact or both divided. The best responses were obtained from the right vagus, and in all experiments vagal stimulation refers to stimulation of the peripheral end of the divided right vagus in the neck, the left remaining intact. Light intratracheal ether anæsthesia was used (with positive pressure when the thorax was opened). In all cases the animals were experimented on twenty hours after their last feed.

**Method 2.**—Secondly, the extrinsic nerves were divided by various operations described below, and later the changes resulting in the œsophagus were studied by means of X-ray plates taken of barium milk introduced by a tube passed from the mouth under ether anæsthesia.

The animals used in all cases were cats. The choice of these animals was made deliberately following the work of Arey and Tremaine[35] on the muscle content of the œsophagus in animals and humans. These observers have shown that these animals correspond more closely to the human in that both possess unstriped muscle in the lower third of the œsophagus, whereas dogs differ from the human in containing striped muscle as far as the cardia, and rabbits are unsuitable in possessing three muscular coats. All experimental results have been confirmed in at least three cases.

## EXPERIMENTAL RESULTS

### METHOD 1

The results obtained from stimulation of the extrinsic nerves were found to vary according to the portion of the œsophagus under examination, and will be described as to vagal and sympathetic stimulation under four zones.

1. **Upper Third of Thoracic Œsophagus.—**

*a. Vagal Stimulation.*—This caused a simple tetanic contraction of the upper third, after a latent period of $\frac{1}{3}$ to $\frac{1}{2}$ second. The contraction passed off immediately stimulation ceased. There were no secondary waves (*Fig.* 111, v). The result appeared to be a shortening of the œsophagus and a straightening of its lower end.

*b. Stimulation of the Stellate Ganglion.*—If carefully isolated, stimulation of this structure caused no contraction of the œsophagus (*Fig.* 111, s s). When, however, this stimulation was combined with stimulation of the vagus there resulted an increase in the degree of contraction of the œsophageal wall (*Fig.* 111, v s). Further, if the vagal stimulation was prolonged sufficiently, until the œsophageal pressure showed signs of returning to the normal level, sympathetic stimulation caused an increase in the contraction above the original vagal rise (*Fig.* 112.)

After prolonged vagal stimulation of approximately 20 seconds followed by an interval of about 15 seconds, sympathetic stimulation alone produced an increase of pressure within the bag (*Fig.* 112, s) which showed oscillations exactly similar to the original oscillations occurring at the crest of the combined vagal and sympathetic stimulations.

2. **Lower Third of Thoracic Œsophagus.—**

*a. Vagal Stimulation.*—This was followed by an increase of tonus and motility in the lower third of the œsophagus, persisting for some time after stimulation (*Fig.* 113). The latent period of this reaction is approximately $\frac{1}{2}$ second; a second type of response consists of a series of secondary waves, three or four in number, occurring at intervals of approximately 10 seconds and following upon an initial contraction or relaxation.

*b. Stimulation of the Stellate Ganglion or Thoracic Sympathetic Chains.*—This caused a slight diminution of tonus and inhibition of the increased motility resulting from vagal stimulation or from mechanical distension of the œsophageal wall by the inflated bag (*Fig.* 114).

3. **Middle Third of Thoracic Œsophagus.**—The effects produced were variable and intermediate between the above.

*a. Vagal Stimulation.*—This caused tetanic contraction followed by secondary waves, a combination of the effects seen in the upper and lower thirds.

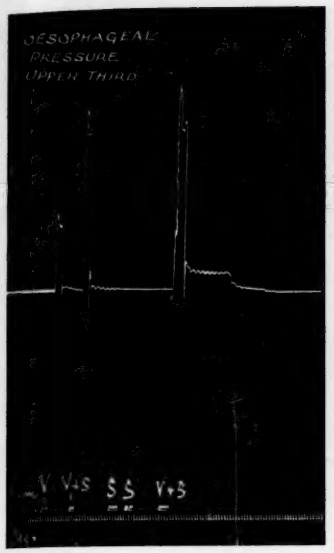


FIG. 111.—Tracing of pressure within the upper third of œsophagus, showing the effects of vagal and sympathetic stimulation, separately and combined.

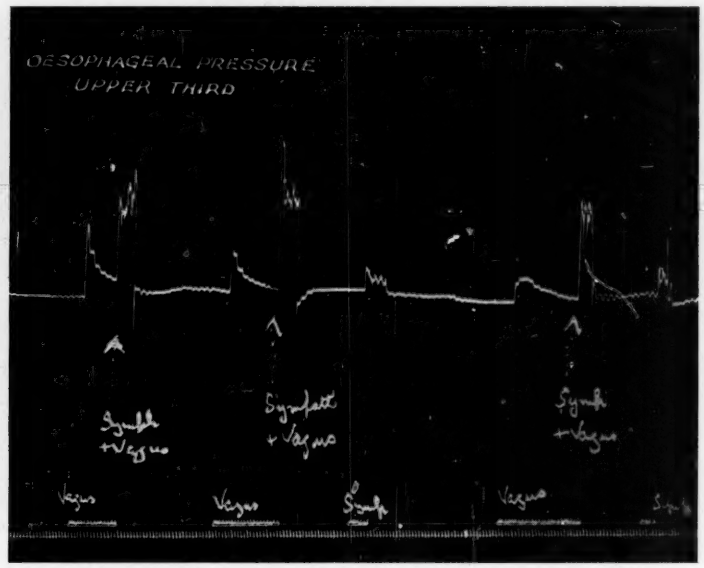


FIG. 112.—Tracing of pressure within the upper third of œsophagus, showing result of sympathetic stimulation upon prolonged vagal stimulation.

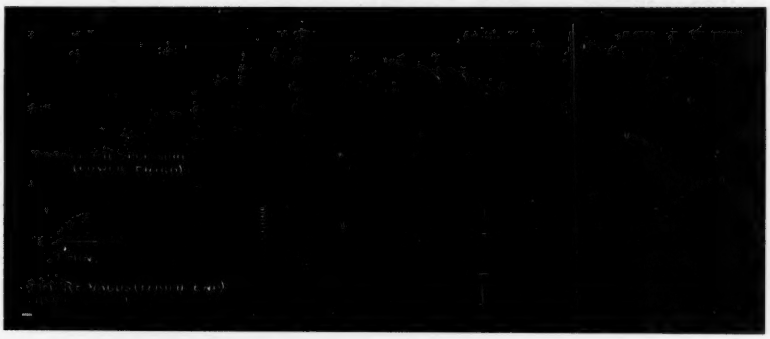

FIG. 113.—Tracing of pressure within the lower third of œsophagus, showing prolonged effect of short vagal stimulation.

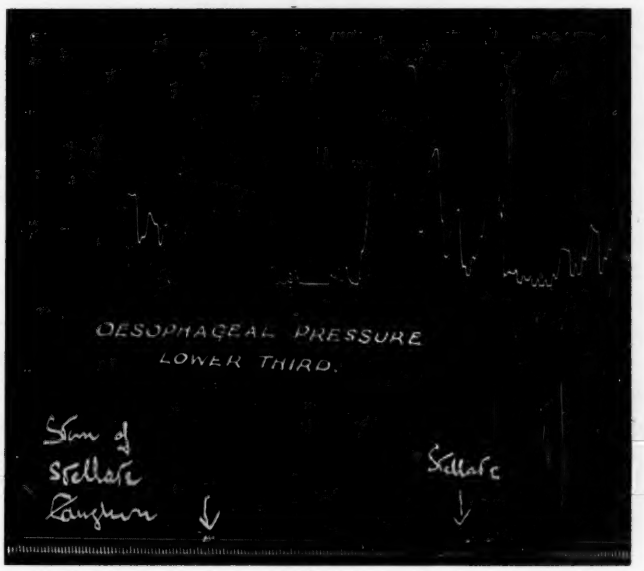


FIG. 114.—Tracing of pressure within the lower third of œsophagus, showing inhibition of motility on sympathetic stimulation.

*b. Sympathetic Stimulation.*—This caused a diminution of tonus or an increase of the contraction resulting from vagal stimulation. The varying results appeared to be due to the exact position of the intra-œsophageal bag.

4. **Inter-diaphragmatic and Intra-abdominal Portion of Œsophagus.**—

*a. Vagal Stimulation.*—This caused immediate relaxation of this portion of the œsophagus in all cases (*Fig.* 115).

*b. Stimulation of the Stellate Ganglion, Thoracic Chains, Splanchnic Nerves.*—No effect (*Fig.* 115, STELL).

*c. Stimulation of the Nerve-fibres surrounding the Cœliac Axis Artery and Left Gastric Artery.*—A gradually increased contraction of this portion of the œsophagus was observed (*Fig.* 116).

Microscopic section of these nerve-trunks showed the presence of medullated fibres among the non-medullated trunks. Reflex effects, such as a complete vomiting reflex and alterations in pulse and blood-pressure, demonstrated the presence of afferent fibres in these trunks. To make sure that the rise of tonus in the œsophageal musculature was not reflex in nature, the experiment was repeated with the artery and nerves divided. Stimulation of the peripheral end still produced a rise in tonus, showing that the effect was not reflex, provided that the strength of the stimulation was increased to compensate for the impaired blood-supply (*Fig.* 117).

The above results suggesting the presence of an intrinsic sphincter at this region, the experiments were repeated with the diaphragm divided, the liver displaced, and the œsophagus lying free. Relaxation was still obtained on vagal stimulation and contraction on sympathetic stimulation (*Fig.* 118).

## METHOD 2

The results of excision of the extrinsic nerves of supply as studied by X-rays of barium introduced into the œsophagus under ether anæsthesia are given below.

**The Normal Cat.**—In studying these plates it is necessary to have for comparison the X-ray appearances of the œsophagus of the normal cat. There is moderate peristalsis of the œsophagus. There is no contraction in the region of the diaphragm and intra-abdominal œsophagus (*Fig.* 119). When the meal has entered the stomach it cannot be returned to the œsophagus by digital pressure exerted upon the stomach contents through the abdominal wall (*Fig.* 120).

**Bilateral Division of Vagi.**—There is dilatation of the thoracic œsophagus and decreased motility, peristalsis recovering later in the lower third. There is contraction in the region of the diaphragm and abdominal portion—i.e., that region which was shown above to exhibit the reactions of an intrinsic sphincter (*Fig.* 121).

If the meal enters the stomach it cannot be returned to the œsophagus by digital pressure (*Fig.* 122).

The degree of the changes produced depends upon the level at which the vagi are divided and the time that has elapsed since operation. *Fig.* 121 shows the condition four months after bilateral division of the vagi below the lung root. Two weeks after operation this animal showed no obstruction at the sphincter. *Fig.* 131 shows the condition in the same animal seven months after operation, in which the obstruction had increased sufficiently to produce vomiting. If the denervation is more extensive and designed to cut off all vagal fibres, by division of the right vagus in the chest below the recurrent laryngeal, and division of the nerve plexus on the

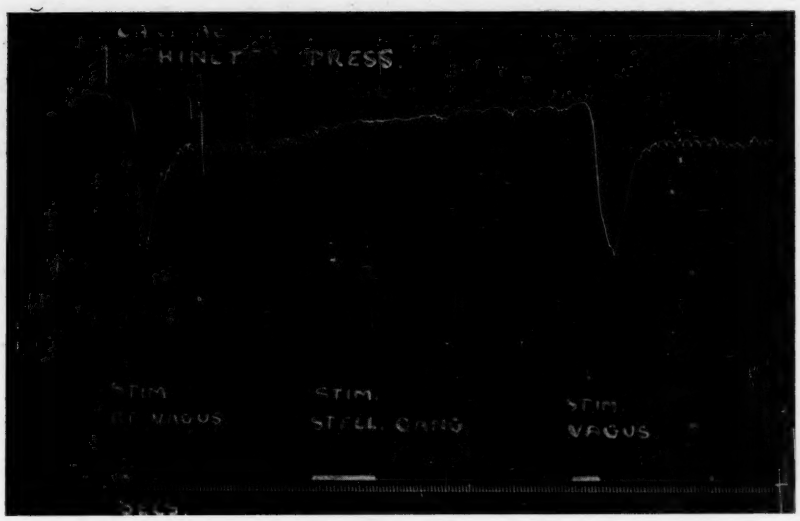


FIG. 115.—Tracing of cardiac sphincter pressure, showing vagal inhibition.

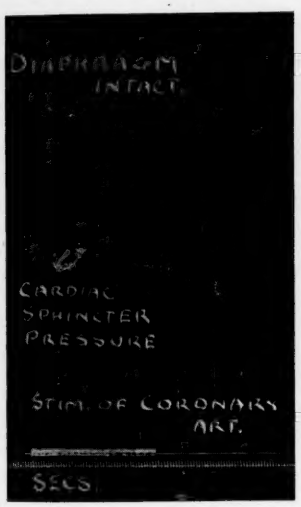


FIG. 116.—Tracing of cardiac sphincter pressure, showing contraction on stimulation of sympathetic nerves on the left gastric artery.

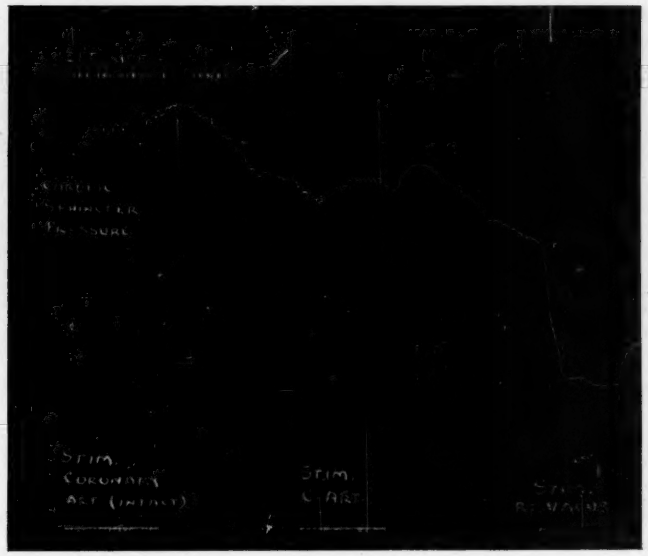


FIG. 118.—Tracing of cardiac sphincter pressure, showing vagal inhibition and contraction on sympathetic stimulation with the diaphragm divided.

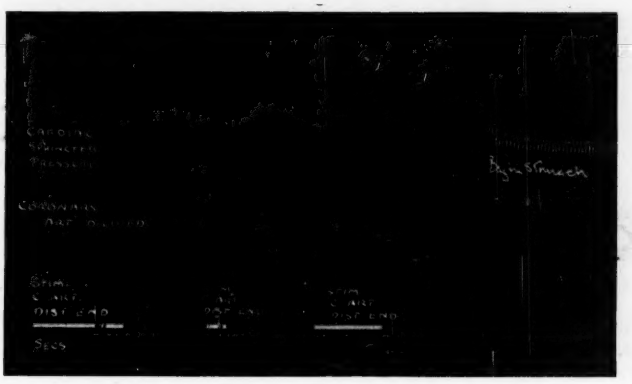


FIG. 117.—Tracing of cardiac sphincter pressure, showing contraction on stimulation of the peripheral end of divided sympathetic nerves.

surface of the œsophagus high in the thorax to interrupt those fibres descending in the plexus from the right recurrent laryngeal, and division of the left vagus in the neck, then the condition shown in *Fig.* 123 is produced. It shows enormous dilatation and an obstruction sufficiently severe to cause death.

**Bilateral Excision of Stellate Ganglia.**—Increased peristalsis is observed in the middle and lower thirds. The sphincter is normal. There is marked hypertonicity in the upper third (striped muscular zone) (*Fig.* 124).

**Bilateral Excision of the Thoracic Sympathetic Chains.**—The third to tenth ganglia were excised in two stages through successive thoracotomies. *Fig.* 125 shows the condition six weeks later. There is a great increase in the peristaltic activity of the œsophagus, it being difficult to obtain a plate of the meal *in situ*. Digital pressure on the abdomen readily returns the meal from the stomach to the œsophagus, showing diminished sphincter tonus (*Fig.* 126).

**Bilateral Division of Vagi at Lung Root, with Bilateral Excision of Thoracic Sympathetic Chains (Ganglia 3-10).**—Performed in two stages through successive thoracotomies. X-ray taken two months later. The œsophagus is at first somewhat dilated and shows decreased motility. The contraction at the diaphragm and intra-abdominal portion does not develop (*Fig.* 127). When the meal has entered the stomach it can be returned to the œsophagus by digital pressure: diminished tonus (*Fig.* 128). After some weeks there is a return of peristaltic activity and the tonus of the sphincter is regained, but obstruction at this region does not occur, the sphincter relaxing readily in the course of peristalsis as in the normal œsophagus.

**Excision of Sympathetic Fibres surrounding the Cœliac Axis Artery.**—The sphincter is completely patulous and cannot support the meal in the vertical position (*Fig.* 129). The meal flows back passively into the œsophagus when the animal is placed in the horizontal position (*Fig.* 130). After some months there is some recovery of tonus, but digital pressure still causes the return of the meal to the thoracic œsophagus.

**Division of the Coronary Artery of the Stomach.**—Changes similar to the above, but less marked, the sphincter being in diminished tonus but not patulous.

**Cœliac Peri-arterial Sympathectomy in an Animal previously treated by Bilateral Division of the Vagi.**—*Fig.* 131 shows the condition of the œsophagus in an animal where both vagi had been divided below the lung root seven months previously. It had been vomiting its food for one week. *Fig.* 132 shows the condition after peri-arterial sympathectomy; the food passes into the stomach without any obstruction. The sphincter is shown in a relaxed condition. There is increased peristalsis of the entire lower third of the œsophagus.

## DISCUSSION

It appears that the type of response obtained from electrical stimulation of the extrinsic nerves passing to the thoracic œsophagus depends upon the nature of the muscle content of the region under examination.

Thus in cats the upper third consists of striped muscle, and in it was obtained the type of response elicited by Kuré and his co-workers[23] from the entire œsophagus in dogs. The striped muscle is prolonged throughout the entire extent

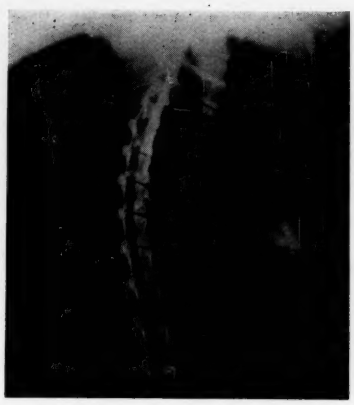

FIG. 119.—X-ray of œsophagus of normal cat, showing no contraction at cardiac sphincter.

FIG. 120.—X-ray of normal cat, showing no return of the meal from digital pressure on the stomach contents.

FIG. 121.—X-ray of œsophagus following bilateral vagotomy, showing contraction at the cardiac sphincter.

FIG. 122.—X-ray following bilateral vagotomy, showing no return of the meal from digital pressure on the stomach contents.

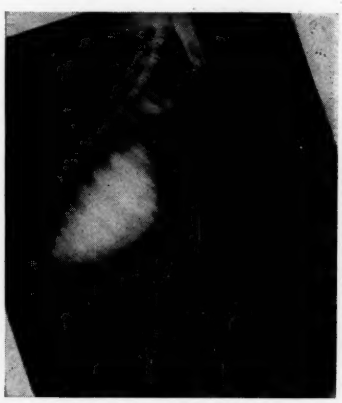

FIG. 123.—X-ray following complete bilateral vagotomy, showing enormous dilatation and complete obstruction at the cardiac sphincter.

FIG. 124.—X-ray following bilateral excision of stellate ganglia, showing hypertonicity of upper third and increased peristalsis. The sphincter was normal.

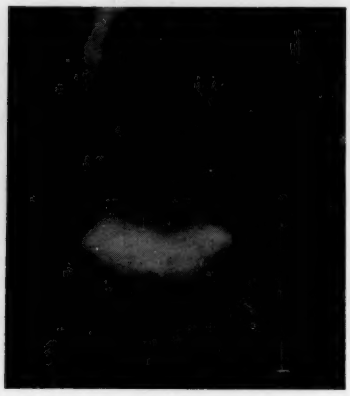

FIG. 125.—X-ray following bilateral excision of thoracic chains, showing increased tonus and motility.

FIG. 126.—X-ray following bilateral excision of thoracic chains, showing the return of the meal from stomach to œsophagus.

FIG. 127.—X-ray following bilateral vagotomy and thoracic sympathectomy, showing absence of contraction at the cardiac sphincter. (Cf. *Fig.* 121.)

FIG. 128.—X-ray following bilateral vagotomy and thoracic sympathectomy, showing return of the meal from stomach to œsophagus.

FIG. 129.—X-ray following cœliac sympathectomy, showing that the meal cannot be supported in the vertical position.

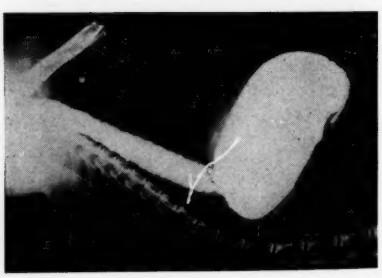

FIG. 130.—X-ray following cœliac sympathectomy, showing passive return of the meal to the œsophagus in the horizontal position.

of the œsophagus in dogs (Arey and Tremaine[35]). Further, the influence on prolonged stimulation is somewhat suggestive of the results obtained by Orbeli[36] upon the influence of sympathetic stimulation on fatigue in skeletal muscle, though differing from this reaction in its immediate and sudden effect.

In the lower third the types of response elicited are those which are associated with the influence of extrinsic nerves upon unstriped muscle elsewhere in the gut —i.e., increased tonus and motility on vagal stimulation and inhibition of motility on sympathetic stimulation. In this region in cats the muscle is predominantly unstriped. The responses also showed a marked similarity to those obtained by Beattie and Sheehan[37] from the stomach in cats, which accorded with the observation of McSwiney[31] that the musculature of the cardia gave a response to drugs which is 'gastric' in type rather than 'œsophageal'. In the middle third variable effects were obtained.

The results obtained from the inter-diaphragmatic and intra-abdominal portions of the œsophagus show the presence in this region of an intrinsic sphincter mechanism, since relaxation was obtained in all cases on vagal stimulation and contraction on sympathetic stimulation.

The changes resulting in the œsophagus from excision of extrinsic nerve confirm the views expressed above. Thus it has been shown that vagal stimulation caused an increase of tonus throughout the œsophagus, with increased motility of the unstriped area and relaxation of the cardiac sphincter. Division of both vagi is followed by dilatation of the œsophagus and contraction of the sphincter. The degree of dilatation and the severity of the obstruction are proportional to the extent of the denervation performed. Immediately following this operation there was also a diminution of peristaltic activity, but this returned after some time as stated by Cannon—i.e., the unstriped area regained the power to respond to local stimuli by local contraction and peristalsis. It is interesting to note that this activity is not accompanied by relaxation of the sphincter, which remains in a condition of increased tonus. It has been stated elsewhere that this obstruction is only temporary in nature—with this we cannot agree. *Fig.* 121 shows the condition in a cat four months after operation. *Fig.* 131 is taken from the same animal three months later, and in this case the obstruction has increased in severity despite the increase in peristalsis.

Stimulation of the sympathetic has been shown to excite certain effects upon the striped muscle of the œsophagus—diminution in the peristaltic activity of the lower third and contraction of the cardiac sphincter. These views again receive confirmation from the results of the operations quoted above. Thus bilateral excision of the stellate ganglia is followed by increased peristalsis in the middle third of the œsophagus. Bilateral excision of the thoracic chains is followed by increased peristalsis and diminished sphincter tonus, and cœliac peri-arterial sympathectomy results in diminution of sphincter tonus.

We were unable to see any sign of the peculiar retropericardial obstruction described by Kuré[24] following bilateral excision of the stellate ganglia, except in so far as hypertonicity of the upper two-thirds caused narrowing of the lumen.

When bilateral vagotomy and excision of the sympathetic chains has been performed, the lower third of the œsophagus has been completely denervated, for the sympathetic supply to the sphincter must take its origin from this source, by whatever route it reaches its destination. In such a case the effects at first observed

appear to be a combination of the above. There is a diminution in tonus and motility of the œsophagus and diminished sphincter tonus. The obstruction which results at this region when the vagi are alone divided does not occur. Later there is a return of peristalsis in the lower third, but this peristalsis is accompanied by relaxation of the sphincter, which does not occur when the vagi alone are divided.

As regards the possibility of the vagus exciting both motor and inhibitory action upon the sphincter, this is not disputed. It is well known that many nerves contain antagonistic fibres which may be selectively stimulated by suitable means. In these experiments contraction occurring at the cardiac sphincter on vagal stimulation has never been observed, but the experiments have in all cases been conducted with high frequency and in animals in the fasting condition. The predominant action of the vagus upon the cardia is inhibitory. In all cases vagus excision has been followed by contraction at the sphincter, and sympathetic excision by diminished tonus.

FIG. 131.—X-ray showing late result of double vagotomy. The obstruction at the sphincter has increased in severity. (Cf. *Fig.* 121.)

FIG. 132.—X-ray showing relief of the obstruction at the sphincter following cœliac sympathectomy. (Cf. *Fig.* 131.)

The bearing of these results upon the etiology of achalasia is interesting. Bilateral division of the vagi results in the clinical symptoms and X-ray appearances of achalasia, this effect being permanent and increasing in severity as time goes on. If the denervation is sufficiently severe, the symptoms are sufficient to cause death of the animal, and the post-mortem appearances of achalasia are produced. *Fig.* 133 shows such a specimen at present in the Museum of the Royal College of Surgeons. It is the thorax of the animal whose radiographic appearances are shown in *Fig.* 123. The œsophagus is enormously dilated—including the upper third—and 'filled the chest', simulating a mega-œsophagus. Rake[38] has shown that in man cases of achalasia show a degeneration of the cells of Auerbach's plexus. These cells Miss C. J. Hill[39] considered to be vagal relays, in which she is in agreement with Gaskell,[20] Kuntz,[40] and Abel.[41]

If, however, the sympathetic fibres are also removed, the obstruction and dilatation do not occur and the œsophagus becomes almost indistinguishable from the normal.

Further, if an animal is prepared so as to reproduce the clinical symptoms and

X-ray appearances of achalasia, subsequent sympathetic denervation of the cardiac sphincter results in a complete cessation of the symptoms, and radiograms show that food now enters the stomach. There is increased peristalsis of the lower third of the œsophagus and relaxation of the sphincter (*see Figs.* 131, 132). There is no increase in the severity of the obstruction such as was observed by Kuré in dogs.[24] Food enters the stomach readily, whole meat being taken, whereas fluid only could be retained before operation. Vomiting ceases and the animal gains weight—in one case to the extent of 0·75 kilos in three months.

As regards the sympathetic innervation of the cardiac sphincter, dissection in human stillborns showed that the thoracic œsophagus derived its sympathetic innervation from two main sources: (1) A few direct branches from the thoracic sympathetic ganglia above the aortic arch; (2) The mesial branches of the ganglia via the peri-aortic plexus and œsophageal vessels. Immediately above the diaphragm no fibres to the œsophagus by either of these routes were seen. The experimental results show that stripping of the fibres surrounding the cœliac axis artery resulted in a patulous sphincter. In the cat these fibres are derived chiefly from the peri-aortic plexus, with some accessions from the cœliac ganglion. Some of these fibres reach the sphincter along the course of the left gastric artery, since division of this vessel results in a diminution but not complete loss of sphincter tonus, such as occurs after cœliac sympathectomy. The splanchnic nerves do not contribute to this supply to any appreciable extent, since no contraction of the sphincter could be produced by stimulation of these structures. This result received confirmation in the human from a case of cœliac sympathectomy performed by Mr. J. B. Hume, Assistant Surgeon at St. Bartholomew's Hospital, for gastric ulcer. Two years after operation the patient was X-rayed and the cardia specifically observed. The sphincter was seen to open abnormally readily, there being none of the usual delay at this region, although actual regurgitation could not be obtained.

FIG. 133.—The thorax of a cat following bilateral vagotomy, showing œsophageal dilatation and obstruction at the sphincter sufficient to cause death. (Cf. *Fig.* 123.)

## CONCLUSIONS

1. The œsophagus receives a sympathetic innervation.
2. There is a true intrinsic sphincter at the cardia.
3. Vagal stimulation causes tetanic contraction of the upper third of the œsophagus, which is composed of striped muscle. This contraction is augmented by sympathetic stimulation.
4. Vagal stimulation causes increased tonus and motility of the lower third of the œsophagus, which is composed of plain muscle. The tonus and motility are inhibited by sympathetic stimulation.

5. Bilateral vagal section, if complete, reproduces the appearances of achalasia of the cardia. Simultaneous removal of the sympathetic fibres prevents the onset of this obstruction. When the obstruction develops it can be relieved by section of the sympathetic supply to the sphincter.

6. The sympathetic supply of the cardiac sphincter is accessible as it passes to the sphincter along the course of the cœliac axis and left gastric arteries.

My thanks are due to Mr. F. Watson, Technical Assistant at the Buckston Browne Farm, for his help in the experimental work, and to Mr. A. Holder, Radiographer at St. Bartholomew's Hospital, for the excellence of his radiograms.

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# THE GROWTH OF PERIOSTEUM IN LONG BONES*

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THE growth of bones at one time appeared a simple matter. Like any other structure in the body they were believed to increase in length and thickness by what may be called *interstitial growth*—a process whereby the increase in size is attained by the growth of each small fragment in length and thickness to a varying extent. The femur of a child bears a close resemblance to the femur of an adult, the astragalus of a child to the astragalus of an adult. The theory that the growth is interstitial afforded a completely satisfactory explanation in each case.

This simple and satisfactory view was shattered by the experiments of John Hunter, who placed pellets at measured intervals in long bones and found that with the growth of the bone the pellets remained the same distance apart. This fact, combined with histological observations by Goodsir of the growing cartilaginous disc near the ends of the bone, established the view that long bones grow in length by linear proliferation of the cartilaginous disc, the so-called *epiphysial or diaphysial line*. As a result of the added complication of this growth disc, many problems of varying importance have been introduced into the puzzle of bone growth.

If a bone grows more or less equally at each end, the original shaft of the child must lie buried in the middle third of the shaft of the adult. If, as has hitherto been assumed, the periosteum also grows at its extremities, it is necessary to find some explanation of the fact that muscle attachments in the adult are not crowded into the middle third of the bone but are arranged in the same relative positions as in the child.

The relation of periosteum to bone growth has been much discussed, but no direct or at any rate conclusive observations have been made on the growth of periosteum itself. One suggestion that presents itself is that no matter how complicated are the processes that obtain in the interior of the bone, periosteum on the outside might conceivably grow interstitially. From an external aspect the growth process in long bones would then bear a close resemblance to that in short or irregular bones (e.g., the carpal and tarsal bones).

The object of the present experiments was to throw light on the behaviour of periosteum in the growth of long bones. It was felt that an understanding of this might help to explain the sliding of muscle insertion and perhaps throw some light on the vexed question of the relation of periosteum to bone limitation and modelling. (The question of the exact constitution of periosteum is left on one side, as it will be seen that our experiments only deal with the fibrous portion.)

* From the Courtauld Institute of Biochemistry.

Rabbits were used for all the experiments. They were usually about six weeks old and were killed after a further six to nine weeks for examination. In every case the tibia of the hind legs was taken as the experimental bone. The insertions of the internal lateral ligament of the knee and of the anterior annular ligament of the ankle were chosen as clearly defined points of attachment of soft tissues to bone.

The experiments may be divided into four groups.

**Group A.**—The first series of experiments were carried out along the original lines of Hunter. The details of these experiments were as follows (*Rabbit* 2, *Fig.* 134):—

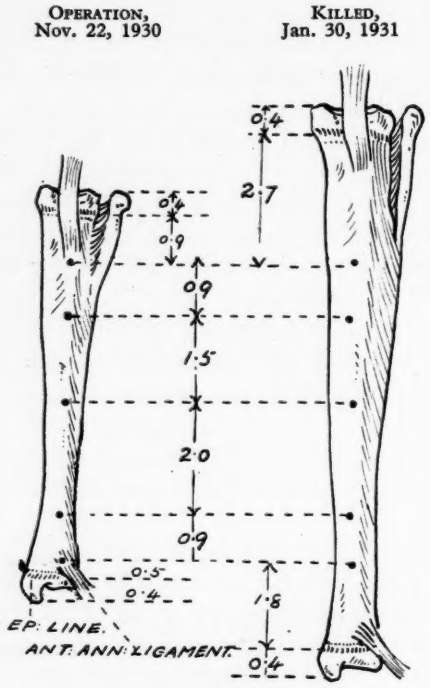



FIG. 134.—*Rabbit* 2.
• = Wire pins.

Silver wire pins were placed in holes drilled through the whole thickness of the bone at measured points which included among others those of the attachments of the internal lateral ligament of the knee and of the anterior annular ligament of the ankle. After nine weeks' growth it is seen that the pins remained the same distance apart, but the insertions of the ligaments had moved considerably from their original sites, indicated by pins. Pinning down the ligaments to the bone has made no difference to this movement.

The results of these experiments are in agreement with those of Hunter. No interstitial growth of bone has taken place. In addition, however, it is to be noted that the attachments of certain ligaments have moved along the bone. Reflection shows that the procedure throws no light on what happened to the periosteum either during the experiments or under natural conditions. Just as the ligaments travel along the bone, so conceivably may the periosteum itself. It may have

grown interstitially or have grown from the end only, or have been prevented from growing by being attached to the underlying bone by the pins.

**Group B.**—It was necessary to mark the periosteum without attaching it to the underlying bone so as to permit it to grow naturally. This presented some difficulty. At first an attempt was made to differentiate growth of periosteum from bone by threading wire loops through the periosteum. It was then found that the part of the loop on the deep surface became incorporated with the bone and remained fixed; although rotation of the loop sometimes occurred, it was obvious that this was not a satisfactory method. Various devices were tried and discarded. Finally, acting on the suggestion of Dr. Scarff, Indian ink was tried, and proved to be quite satisfactory.

The bone was exposed with the usual surgical aseptic technique and the periosteum carefully dried with swabs. At suitable intervals marks of Indian ink

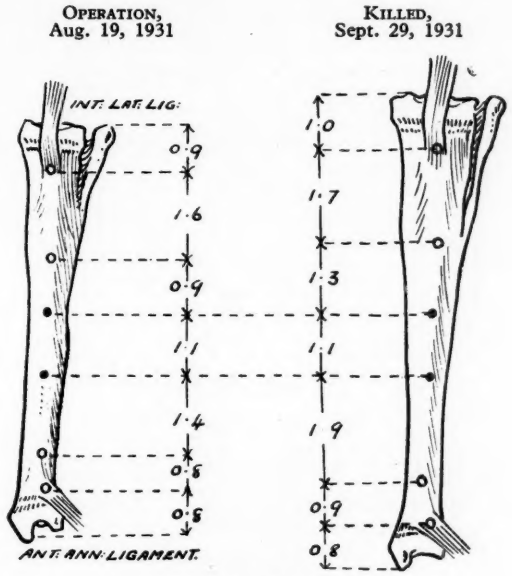



Fig. 135.—*Rabbit* 6.
• = Wire pins. o = Indian ink.

were then made superficially in the substance of the periosteum with a mapping pen. Dividers were used for making all measurements, which are recorded in centimetres. At the end of two months the marks, with a few exceptions, were still clearly visible, although they had become slightly fainter and more diffuse. Owing to the impossibility of making extremely small marks, the measurement between any pair of marks can only be considered accurate to the nearest millimetre.

Metal pins placed through the diaphysis of the bone remain unchanged in position and can be regarded as fixed points from which to make future measurements. Two pins were placed through the bone to act as fixed points and ink marks were made on the periosteum and ligaments. If the periosteum grew only by increments from its ends, then the positions of the ink marks relative to the pins should remain unchanged. That this does not occur is very clearly shown by *Rabbit* 6 (*Fig.* 135).

Clear evidence was thus obtained that interstitial growth of periosteum takes place. Ink marks placed in the ligaments at their insertion were found still to remain at the junction of ligament and periosteum.

**Group C.**—Further experiments showed that the periosteum adjoining the ligamentous attachment changed its position with that of the ligament itself. Ink marks were made on the ligaments and the periosteum at exactly the same level. The relative positions of these two marks were unchanged when the animals were

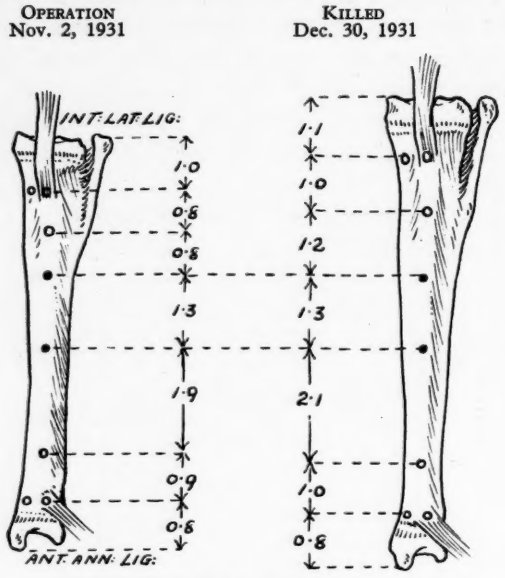



FIG. 136.—*Rabbit* 10.
• = Wire pins. o = Indian ink.

killed (*Rabbit* 10, *Fig.* 136). It is therefore unlikely that the drag of the shifting of ligament insertions plays much part in the separation of given points in the periosteum.

It has thus been shown that the insertion of a ligament always preserves the same relationship to the fragment of periosteum to which it was originally attached. In other words, the two halves of a mark which at any period indicates the junction of the periosteum with any given ligament remain undivorced during growth of the bone, and presumably the same also holds good for the relationship between periosteum and any muscle insertion.

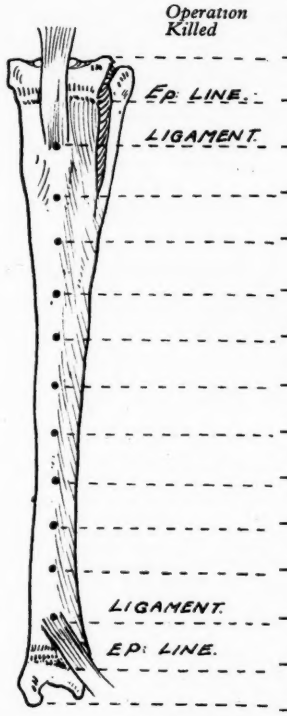
It would have been of interest in this connection to observe any change in the position of the lower tibio-fibular synostosis relative to the points fixed by the pins. However, it was not thought advisable to attempt to make this observation at the same time from fear of damaging periosteal growth.

It is now necessary to consider briefly how the ligamentous insertion changes its position. If the ligament is merely attached to the periosteum itself, no difficulty arises, as the ligament simply moves with the growth of its related periosteum. The solidity with the bone beneath must then be brought about by the rapid fixation of fresh periosteal perforating fibres formed in the subperiosteal plane in response to the stress on the slowly shifting insertion.

It is generally held, however, that "where tendons or ligaments are inserted into bone, the fibre bundles of the tendon are continued into the bone as perforating fibres, so that the attachment of tendon to bone is thus rendered very intimate" (Schäfer[1]). If this view is accepted, the perforating fibres must be supposed to elongate as the ligament shifts its position, for they cannot be supposed to re-insert themselves higher up the bone. The elongation may take place in the bone itself or in the part of the fibres which runs immediately under the periosteum. The latter suggestion is more likely. It may then be supposed that the freshly formed periosteal bone fixes the fibres in their slowly changing position. The subperiosteal changes are not affected by transfixation of the ligament to the bone by pins.

No matter what view of these fibres is taken, the effect produced is that the insertion of ligament and probably of muscle with the associated pits and ridges marking these insertions must actually travel subperiosteally along the bone as it grows.

**Group D.**—Finally an attempt was made to discover if the increase in length of the periosteum took place by regular interstitial growth—that is to say, as evenly as a piece of elastic stretches—or if it were more irregular. To this end a series




| Operation / Killed | Rabbit 15<br>Jan. 28, 1932<br>March 9, 1932 | | Rabbit 14<br>Jan. 21, 1932<br>March 9, 1932 | | Rabbit 13<br>Jan. 20, 1932<br>March 9, 1932 | |
|---|---|---|---|---|---|---|
| | 0·4 | 0·4 | 0·4 | 0·4 | 0·3 | 0·3 |
| | 0·5 | 0·5 | 0·5 | 0·7 | 0·5 | 0·8 |
| | 0·5 | 0·5 | 0·5 | 1·1 | 0·5 | 0·6 |
| | 0·5 | 0·6 | 0·5 | | 0·5 | 0·6 |
| | 0·5 | 0·6 | 0·5 | 0·7 | 0·5 | 0·6 |
| | 0·5 | 1·3 | 0·5 | 0·6 | | |
| | 0·5 | | 0·5 | 0·5 | | |
| | 0·5 | 0·7 | 0·5 | 0·5 | | |
| | 0·5 | 0·6 | 0·5 | 0·8 | 0·5 | 0·7 |
| | 0·5 | 0·7 | 0·5 | 0·9 | 0·5 | 0·7 |
| | 0·5 | 0·6 | 0·5 | 0·7 | 0·5 | 0·7 |
| | 0·5 | 0·6 | 0·5 | 0·6 | 0·5 | 0·8 |
| | 0·5 | 0·5 | 0·4 | 0·4 | 0·4 | 0·6 |
| | 0·4 | 0·4 | 0·4 | 0·4 | 0·3 | 0·3 |
| Total length | 6·8 | 8·0 | 6·7 | 8·3 | 5·0 | 6·7 |

Fig. 137.

of ink marks was made on the periosteum of the entire length of the shaft at intervals of 0·5 cm. After growth it was found that, with few exceptions, each interval was increased in size, although not all to the same extent. Occasionally isolated ink spots could not be found. (*Fig.* 137.)

## SUMMARY AND CONCLUSIONS

1. The growth of the periosteum of the tibiæ of young rabbits was observed by means of marks of Indian ink.

2. It was found that periosteal growth did not occur as in the underlying bone by increments from the epiphysial lines, but by interstitial growth throughout its length.

3. The limited number of observations that were carried out indicate that the interstitial growth is reasonably regular. The results are recorded.

4. The process of ligament and muscle sliding during normal growth of long bones, and possibly also the limiting of growth and modelling of the bone, are related to the interstitial growth of periosteum.

We wish to express our indebtedness to Professor Dodds for the hospitality of the Courtauld Institute of Biochemistry, in which the work was carried out. Our thanks are due to Mr. E. R. E. Spence, technician, for his assistance and care of the animals.

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[1] SCHÄFER, *Quain's Anatomy*, 1912, ii, Part 1, 151.

# *SHORT NOTES OF RARE OR OBSCURE CASES*

## FALLOPIAN TUBE IN THE MALE: DISCOVERED WHILE OPERATING FOR FEMORAL HERNIA

By J. A. BATY

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A MINER, aged 29, attended the Out-patient Clinic of the Cumberland Infirmary, complaining of "rupture" in the right groin. He stated the rupture had been present since birth. The swelling always disappeared spontaneously when he lay down. There had never been symptoms of threatened obstruction. His own doctor had attended him a few months previously for an attack of left-sided epididymitis which lasted about a fortnight. There has been a transitory yellow, opaque, sticky discharge, but no other urinary symptom. No evidence of gonococcal infection had been discovered. The patient had been married over a year. Marital relations were apparently normal, but birth control had been practised and there was no child.

ON EXAMINATION.—The patient was well developed and had a rather pugilistic type of face. All the secondary male characteristics were in evidence. No testis could be felt in the right side of the scrotum, inguinal canal, or femoral triangle. There was a femoral hernia which could easily be reduced. On the left side the testis was of average size with a definite corpus testis and epididymis, and lay in the scrotal sac. The cord was normal and there was no detectable hernia. Per rectum, the prostate was found to be very small, globular, and no larger than a pea.

OPERATION (Nov. 30, 1933).—Operation for right femoral hernia was performed by Mr. J. N. J. Hartley. The inguinal canal was first explored. There was no sign of the testis, cord, or hernial sac in the canal. The femoral hernial sac was then dissected free and brought up into the inguinal canal. It was about the size of a thumb and when opened the neck readily admitted the index finger. A search was made for an undescended testis, but no testis could be felt by the finger. While retracting the neck of the sac, the free extremity of a tube appeared at the inferomedial margin. This was gently brought out, and to our surprise it had all the appearance of a miniature Fallopian tube. By further traction the tube was seen to be continuous with a firm muscular structure about the size of the distal phalanx of the little finger. The opposite angle of this body could not be reached and it was therefore not determined whether a tube existed on the left side.

The mesosalpinx and the inner end of the tube were ligated, and the tube was removed for investigation. The neck of the sac was ligated by a purse-string suture applied from within, and the hernial sac pursed up by a catgut suture. The femoral walls were narrowed by non-absorbable sutures and the wound was sutured.

DESCRIPTION OF THE TUBE.—The excised tube is about 3 in. in length, and, as will be seen from the accompanying sketch (*Fig.* 138), possesses a fimbriated extremity with a distinct ostium. The tube is lying along the free upper border

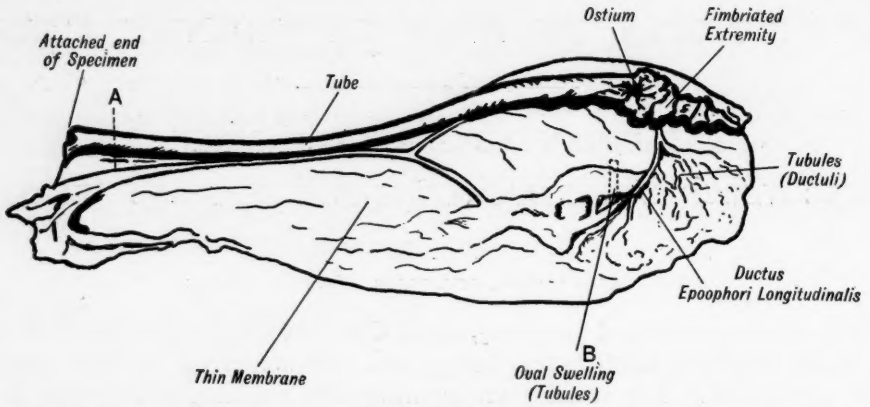


FIG. 138.—Specimen of Fallopian tube removed from male patient. Drawing of specimen from posterior aspect. A and B indicate source of sections for microphotographs.

of a thin membrane on the posterior aspect of which a number of rudimentary structures can be seen. Passing out from under cover of the fimbriated end of the tube is a definite ridge which divides into two main stems forming a triangular

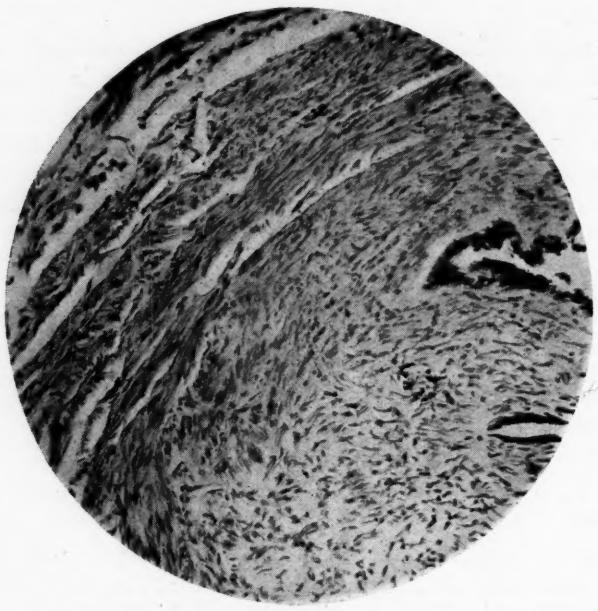

FIG. 139.—Section taken from A in *Fig.* 138. Fallopian tube. Low-power view showing structure. For details *see* text.

area occupied by smaller radiating ridges. These, at the periphery, anastomose. Just above the medial main ridge there is a flattened boss about $\frac{1}{2}$ in. by $\frac{1}{4}$ in. and below the latter are further ridges, branching and rejoining. From the medial end

of this system a single ridge arches up towards the tube and for a short distance runs parallel along the latter's lower border. Separation of these structures again occurs at a point about ¾ in. from the attached end of the specimen.

*Histological investigation* (*Fig.* 139) shows the tube to possess a tunica serosa separated by a tunica adventitia of loose connective tissue from a tunica muscularis. The latter is composed of two strata of smooth muscle fibres, the outer longitudinally arranged and the inner circularly. The submucosal layer of embryonic fibroblastic cells supports the mucous membrane, which is thrown into a number of longitudinal folds. The lining epithelium consists of columnar cells with large, pale central nuclei showing a fine chromatin network. The free surface presents small protoplasmic protusions which are definitely cilia.

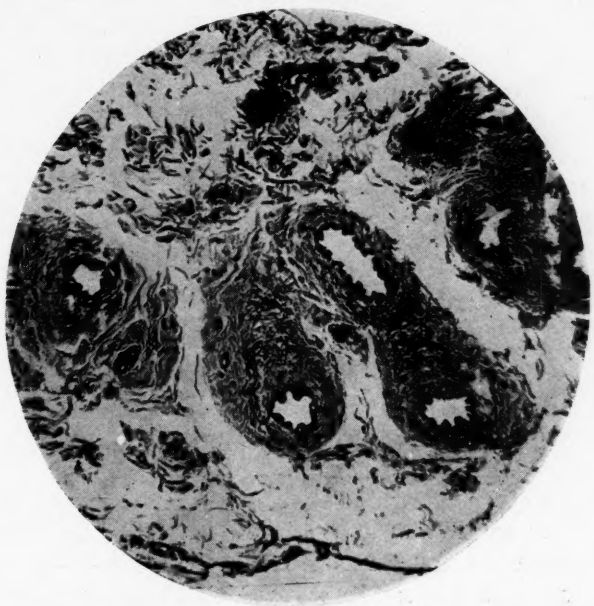

FIG. 140.—Section taken from B in *Fig.* 138. Tubules. Low-power view showing structure of oval swelling below fimbriated extremity of tube.

A section taken to include the ridges and oval swelling in the membrane below the fimbriated end of the tube reveals no evidence of ovarian or testicular tissue. For the greater part this area is composed of small tubules lined by ciliated epithelium and held in apposition by loose connective tissue (*Fig.* 140).

## COMMENTARY

There can be no doubt that in this patient there existed a diminutive but well-developed Fallopian tube. The Müllerian element on the right side has become dominant. There are vestiges of the Wolffian derivative, and these are more in evidence than in the normal female. The epoophoron with its ductus epoophori longitudinalis can readily be distinguished. The ductuli correspond to the efferent ductules of the testis, but no spermatocytic cells are present. The ductus epoophori longitudinalis is the remains of the Wolffian duct and is the homologue of the vas in the male and the duct of Gärtner in the female.

The embryological explanation for the developmental abnormality can only be conjectured. Undoubtedly sex differentiation rests upon chromosomal constitution: it is determined at the moment of union between sperm and ovum according to whether the sperm carries the X or Y chromosome. Before sex differentiation becomes reinforced by endocrine activity, the chromosomal influence appears so weak that the developing embryo passes through an indifferent phase, structures proper to both male and female making their appearance. This phase is followed by a masculinizing tendency in both sexes. In the female a testicular rudiment appears in the rete of the ovary, the investing ovarian cortex becoming the real ovary. In the male the tendency is evidenced by the earlier differentiation of the testis. It is held that the testis is a monosexual organ, whilst the ovary is bisexual, and that sex reversal can only occur in the female. Despite the recent work on accidental and experimental sex reversal, it would be hazardous to assume in the human subject with a testis so well developed that the chromosomal content was other than male. The rudimentary excretory apparatus when the sex gland is absent calls for no comment. It is not clear why the Müllerian element should develop to such an extent in a male, for mere absence of one testis does not lead to unilateral preponderance of female organs. Until there is a more exact knowledge of the sexual hormones and their actions, the true explanation of this case will remain in doubt.

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## A RARE ABNORMALITY OF THE HEPATIC ARTERY

By G. A. G. MITCHELL

ASSISTANT IN SURGERY, UNIVERSITY OF ABERDEEN

Abnormalities of the cystic and hepatic arteries and of the biliary apparatus are well recognized and their surgical importance has been frequently emphasized, but it is doubtful if many surgeons realize that the presence of an abnormal cystic artery not infrequently indicates an abnormality of the parent hepatic stem. The hepatic artery and its branches conform to the text-book description in only 50 to 55 per cent of cases. In the remainder abnormalities of the most varied character may be encountered, some relatively common, others comparatively rare. The discovery of one of the latter in a male subject aged 63 prompts me to give the following details.

The abdominal aorta was normal and the branches came off in the usual situations, but the cœliac axis was smaller and the superior mesenteric artery larger than usual. The coronary and splenic branches of the cœliac axis conformed to the usual arrangement, but the hepatic artery was very small, and its only branch was a tiny twig to the pyloric region of the stomach; it gave off no other macroscopic branches before dividing into minute vessels which entered the liver.

Arising from the superior mesenteric artery in front of the uncinate process of the pancreas was a vessel which was as large as the continuation of the artery itself. It ran upwards in the *anterior* groove between the duodenum and the head of the pancreas, then behind the first part of the duodenum, and finally in the

free margin of the lesser omentum to the right of the common bile-duct, which thus had an artery on each side and the portal vein behind it. This abnormal branch gave origin to the middle colic, right gastro-epiploic, pyloric, and cystic arteries (*Fig.* 141), and supplied also those structures usually supplied by the pancreatico-duodenal vessels. Finally it entered the portal fissure, its two terminal branches lying anterior to the hepatic ducts. The cystic artery lay below and to the right of the cystic duct. The biliary apparatus and the other viscera were normal, and there was no indication that the abnormal artery was produced by

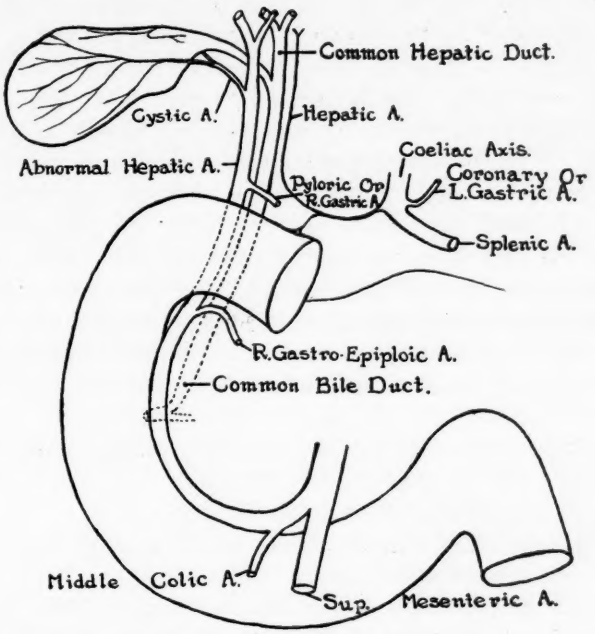


FIG. 141.—Abnormality of the hepatic artery.

post-natal enlargement of the anastomoses normally present between radicles of the pancreatico-duodenal arteries.

The appearances suggested that the major part of the liver, the biliary apparatus, the pyloric end of the stomach, the greater part of the duodenum, the head of the pancreas, and the transverse colon received their arterial blood-supply through this curious vessel. Any injury, operative or otherwise, to such a vessel would be fraught with the gravest consequences, and from its situation it would be endangered in most operations in this region.

In his exhaustive "Essai sur l'Anatomie et la Médecine opératoire du Tronc cœliaque et de ses Branches" Rio-Branco discusses abnormalities of the hepatic artery, but of the large number of abnormalities he describes there is not one exactly similar to the above; the nearest approach to it is a case reported by Wiart. Moreover, in the cases described in the literature I have not found any in which so many structures were so largely dependent on the abnormal artery.

The origin of an abnormal hepatic artery from the superior mesenteric artery is a well-known possibility, but almost invariably it arises near the beginning of

the superior mesenteric, *behind* the pancreas, and then passes upwards and to the right, behind the first part of the duodenum, towards the free margin of the lesser omentum, where it may lie to the right or to the left of the common bile-duct or posterior to it. In one case described by Corning the abnormal vessel, although arising behind the pancreas, passed *anterior* to the first part of the duodenum. In most of these abnormal cases another hepatic artery exists, arising as usual from the cœliac axis or directly from the abdominal aorta, or less commonly from the coronary or splenic arteries. This artery may be larger or smaller than the one arising from the superior mesenteric, and it is sometimes called the 'left' or 'superior' hepatic artery, in contradistinction to the other, which is described as the 'right' or 'inferior' or 'accessory' hepatic artery; the term 'accessory', however, is misleading, because in many instances the right or inferior artery is the larger of the two. They are designated 'right' and 'left' because in their final distribution they usually conform more or less closely to the right and left terminal branches of the normal hepatic artery, but Corning, Leriche, and others record cases where the so-called left or superior hepatic artery ended in the right lobe of the liver. In other cases three hepatic arteries are present, which frequently arise as follows: the first from the coronary or less often from the pyloric artery, the second from the cœliac axis or directly from the abdominal aorta, and the third from the superior mesenteric artery or from a combined cœliaco-mesenteric trunk. Isolated cases have been recorded of abnormal hepatics arising from, or in conjunction with, the renal or inferior phrenic arteries, and from the inferior mesenteric artery.

In 338 subjects examined by Leriche and Villemin, Sousloff, Rossi, and Cova, and by Rio-Branco, the hepatic artery arose from the cœliac axis in 315 cases, from a combined cœliaco-mesenteric trunk in 4 cases, from the superior mesenteric artery in 14 cases, and directly from the aorta in 5 cases. Thus in 4 per cent of cases an abnormal hepatic artery may arise from the superior mesenteric artery. It is worth emphasizing again that in the great majority of these cases the artery originates and pursues a relatively safe course behind the pancreas and first part of the duodenum, but in the specimen described above the abnormal hepatic arose anterior to the pancreas and pursued a totally different course. The latter abnormality is comparatively rare, and from a surgical point of view much more dangerous.

I am indebted to Professor Low, Department of Anatomy, Aberdeen University, for kind permission to publish this case.

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# DISLOCATION OF THE ELBOW WITH RUPTURE OF THE BRACHIAL ARTERY

By RALPH MARNHAM
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On May 16, 1933, Mrs. M., a woman aged 40, fell from a step-ladder, a distance of some four feet. She was able to describe the accident accurately. She fell on to her left hand with the arm extended, the forearm fully supinated, and the hand dorsiflexed.

On Examination.—When seen an hour after the accident she complained of pain at the elbow-joint and tingling of all her fingers. There was extreme swelling in the region of the elbow-joint, and in the absence of an X-ray it was impossible to assess the exact nature of her injury. She had no signs of damage to nerves, but the radial pulse was absent.

Operation.—It was decided that she should be operated upon immediately. An incision 4 in. long was made over the anterior aspect of the internal epicondyle. On dividing the skin a large blood-clot was immediately evacuated. When the edges of the incision were retracted only two structures were seen running across the front of the elbow-joint; the one was the tendon of the biceps, the other the median nerve. All the muscles had been torn off the internal epicondyle and the brachial artery had been divided about $\frac{1}{2}$ in. above its bifurcation. There was a firm clot about $\frac{1}{4}$ in. long in the proximal lumen. The radius and ulna were dislocated backwards without any fracture; this was subsequently confirmed by an X-ray examination. The dislocation was reduced and the proximal end of the brachial artery ligatured; the distal end of the brachial artery had retracted, but was found and ligatured. The torn muscle was approximated by mattress sutures. After the skin incision had been closed, the arm and hand were covered with several layers of cotton-wool and kept warm with hot bottles. The arm was placed on a pillow in about 20° of flexion and in full pronation.

Subsequent Progress.—The next day the arm and hand were warm and sweating. The radial pulse had reappeared by the end of three weeks. By the end of four months extension of the elbow-joint was limited by about 10°; otherwise movements were full. There was no noticeable loss of power in flexing the forearm. The patient never had massage or passive movements.

The points of interest in this case are: (1) The rarity of the condition—I have been able to discover only one other case recorded in the literature[1] for the last thirteen years; (2) That with trauma sufficient to rupture not only the brachial artery but also the flexor muscles, the nerves, particularly the median, and the collateral circulation should have remained undamaged.

## REFERENCE

[1] *Jour. de Méd. de Bordeaux*, 1917, July, 522.

# LARGE PREVERTEBRAL HÆMATOMA CAUSING PARALYSIS OF THE RECURRENT LARYNGEAL NERVE

By CECIL P. G. WAKELEY
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RICHARD S., age 22, a tyre fitter, was admitted to King's College Hospital on Nov. 21, 1933, complaining of a very swollen neck and difficulty in swallowing and speaking. Half an hour before admission, while trying to lever a motor tyre on

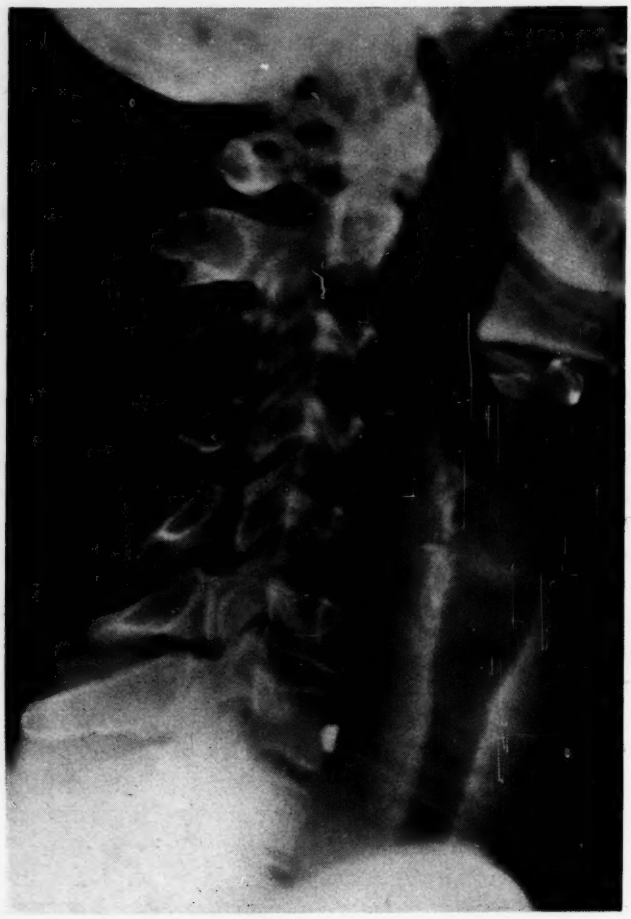

FIG. 142.—Skiagram showing foreign body in the 7th cervical vertebra.

to the wheel of a lorry, a small piece of the tyre lever was chipped off and pierced his neck. The wound of entry was very small and there was but little bleeding.

On examination a small wound was seen about $\frac{1}{4}$ in. in diameter and $1\frac{1}{2}$ in. above the left sternoclavicular joint. A lateral X-ray revealed a foreign body about $\frac{1}{4}$ in. long situated in the anterior part of the body of the 7th cervical vertebra,

a little to the left of the mid-line. The skiagram also shows the track of the piece of metal as it passed through the fascia of the neck (*Fig.* 142).

Examination of the interior of the larynx revealed paralysis of the left vocal cord.

The patient was admitted to hospital as his condition did not improve. The next day the wound of entrance was excised and the wound explored. With a bullet probe the piece of metal could be felt firmly embedded in the vertebra, but as the exploration caused considerable bleeding it was deemed wise not to proceed further. The large hæmatoma gradually subsided and the swelling ceased to be painful. On examination with the œsophagoscope a large bulge could be seen on the posterior pharyngeal wall. The paralysis of the left recurrent laryngeal nerve gradually cleared up, and by the end of three weeks there was no sign of any involvement of this nerve, and the patient was discharged.

This case is of interest because of the curious nature of the injury and the large size of the prevertebral hæmatoma which caused paralysis of the recurrent laryngeal nerve. The piece of metal must have taken a course very near the common carotid artery, as it must have pierced the carotid sheath.

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## BILIARY CYST OF THE LIVER*

By E. ROCK CARLING

SURGEON, WESTMINSTER HOSPITAL

B. J., female, aged 28, was admitted to Westminster Hospital in July, 1927. For six years she had observed an enlargement of the veins on the right side of the trunk, and latterly they had become very much larger; she was troubled by a dull aching pain on the right side and had consulted Dr. Alment, of Abbot's Langley.

On Examination.—The veins extended from the umbilical region up over the chest, mainly towards the right breast and axilla, but partly also over the lower part of the sternum. The blood flowed upwards in the umbilical, superior epigastric, lateral axillary, and mammary vessels. Close to the umbilicus a definite thrill was palpable in the dilated veins. The liver projected about two finger-breadths below the costal margin and could be felt on careful palpation.

Upon radiographic examination the diaphragm was seen raised to the level of the 3rd rib. There was no marked displacement of the heart. The radiologist suggested that there might be a tumour between the liver and diaphragm.

The temperature ran from 98° to 99° in the evening; the urine was normal; bowels freely open. Hæmoglobin, 75 per cent; red blood-cells, 4,770,000; white blood-cells, 5600; Wassermann reaction, negative; cholesterol, 110 mgrm. per 100 c.c.; bilirubin, 0·1 units; lævulose tolerance, normal. No hæmorrhoids. No symptoms of importance.

Operation.—After the case had been shown at 'consultations' it was decided to explore. The right lobe of the liver was occupied by a large cyst which came

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* For a bibliography of non-parasitic cysts of the liver, *see* the article by Ackman and Rhea, British Journal of Surgery, 1931, xviii, 648.

to the hepatic surface just lateral to the gall-bladder and was there thin-walled. It was incised, and a large quantity (2½ pints) of golden yellow liquid with quantities of cholesterol evacuated. Bile pigments and salts were present. After emptying, the cavity easily admitted the whole hand and wrist, so that it was possible to feel and compress the inferior vena cava at the back of the liver. There was no 'cyst wall' separable from liver substance over the greater part of the parietal area and very little collapse of the liver substance, so an anastomosis was made with the greater part of the adjacent gall-bladder, using the same technique as for joining two hollow viscera.

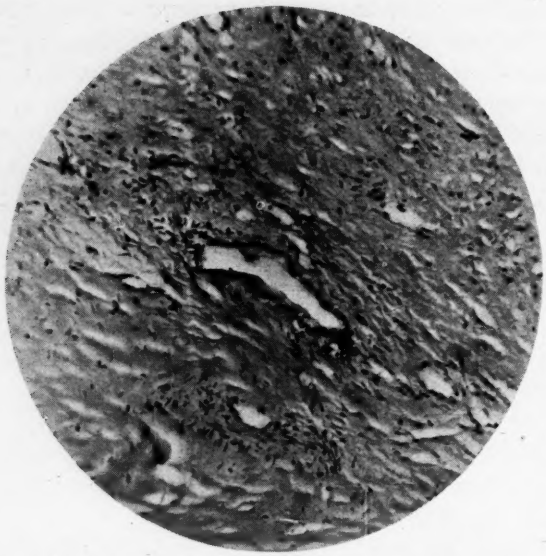

FIG. 143.—Histology of the biliary cyst of the liver.

Recovery was without other incident than the formation of a sinus that healed after removal of a ligature. In a month's time the diaphragm, though still at the level of the 4th rib, was now freely mobile, whereas before the operation it had been stationary. The veins were perceptibly smaller two days after operation and gradually disappeared. There was a slight excess of cholesterol in the fæces. The cyst wall showed a hyaline fibrous matrix with very thick walls, with small spaces lined by cuboidal cells resembling those of the biliary ducts (*Fig.* 143).

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## LARGE SOLITARY CYST OF RENAL ORIGIN

BY E. ROCK CARLING

SURGEON, WESTMINSTER HOSPITAL

IN the year 1911 Mrs. E. B., aged 47, was sent for consultation by Dr. Anderson, of Alleyn Park. She was preceded into the room by her husband, who walked backwards, supporting his wife's abdomen with both hands. Without such help she could scarcely stand, and certainly not walk, so great was the bulk and weight of the cyst.

Her general health was fairly good and she had no symptoms other than those due to the size of the abdomen. Micturition and the urine were normal. There was no jaundice. Though thin, she was not emaciated; she took food in small quantity but regularly; the bowels were regular; catamenia regular, but lately a little excessive.

She had allowed herself to suffer disabling discomfort, because, so she alleged, the most highly distinguished gynæcologist of that time, who had seen her in 1896, fifteen years before, had advised her never to allow any surgeon to operate upon her—this although he had diagnosed an ovarian cyst.

On Examination.—The entire abdomen seemed to be filled with fluid, presumably contained within limiting walls, but it was impossible to identify any organ. The stomach and intestines seemed to be on the left side, but the only area of resonance obtainable was very small in extent; the pelvis was full of the 'cyst', which was thought (in view of the earlier diagnosis), although it was quite immobile, to be of ovarian origin.

Operation.—An incision, which was meant to be paramedian, exposed the wall of the cyst without traversing the peritoneum. Upon incision twenty-five pints of fluid were collected and much was lost. The fluid was odourless and turbid. The turbidity was due in the main to cholesterol, which was present to the amount of 4 lb.; there was also a considerable amount of squamous epithelium; the reaction was amphoteric; there was in this fluid no reaction for urea.

The entire peritoneal cavity and all the viscera had been pushed over to the extreme left. With the hand inside the cyst, the aorta and spinal column could be closely palpated. A renal origin seemed probable at this stage of the operation, though something that seemed to be the normal kidney could be palpated.

An attempt to enucleate the cyst resulted, whilst the dissection was still being conducted well above the collapsed abdominal wall, in damage to the posterior wall of the duodenum, fortunately not opening the mucosa. As much as possible of the cyst wall was excised, and the cavity then marsupialized.

Subsequent History.—The discharge, three weeks later, had a specific gravity of 1030; effervesced with sodium hypobromite; contained squamous, pyriform, and spheroidal cells and triple phosphates; it did not digest albumen or form sugar from starch. The discharge ceased before the patient left hospital, but re-opened at times up to 1915, when she was lost sight of. In 1912 the patient returned to hospital for a hysterectomy for fibroids, which had increased in size with great rapidity in the intervening fifteen months.

**Commentary.**—It is possible that this was not a 'cyst' in the true sense, but a hydronephrosis into which a hæmorrhage had occurred, but the mass of renal substance palpated through the wall seemed normal, and at the time in question pyelograms were not in use.

The urine passed had a specific gravity of 1018 and contained oxalates; in the fluid from the sinus, which contained very small quantities of urea, there were triple phosphates, and the pathologists, Dr. Hebb and Dr. Braxton Hicks, believed that there was at least a connection with the renal pelvis. As some pararenal cysts have such a connection, this extreme example should probably be so classed.

# MALIGNANT DISEASE STARTING IN A PHARYNGEAL POUCH

By DUNCAN C. L. FITZWILLIAMS
SENIOR SURGEON, ST. MARY'S HOSPITAL

In the British Journal of Surgery for April, 1933, Capp and Dunhill published the details of a case of malignant disease which had started in a pharyngeal pouch before operation. Owing to the rarity of such a happening, perhaps the following case where malignant disease started in a pharyngeal pouch after operation may prove interesting.

W. R. H., a man aged 55, was admitted to the Freemasons Hospital in January, 1928.

History.—For two to three years he had experienced difficulty in swallowing, which had gradually increased till he was unable to eat solids. His food in part regurgitated unchanged with no acid taste. There was no loss of weight.

On Examination.—He was a short man with a thick neck; there was fullness above each clavicle, but this could not be felt to increase on either side on eating.

Report of the X-ray examination: The first mouthful swallowed was held up at the level of the top of the sternum and shows as a smooth rounded mass. The next mouthful passed fairly easily down the œsophagus, flowing past the upper portion of the previous swallow. These points indicate an œsophageal pouch.

First Operation.—At the operation I was assisted by my colleague, the late William Hill. An incision was made on the left side of the neck, the sterno-mastoid muscle was defined and drawn forward, the omohyoid was divided. A bougie was now passed into the pouch through the mouth, and the pouch defined, freed, and brought to the surface. It was very thin, consisting only of mucous membrane. Fine catgut sutures were inserted into the free end of the bag, which was displaced upwards and attached to the fibro-fatty tissue at the lower pole of the parotid underneath the superior thyroid vessels. The omohyoid was joined up and the wound closed with a drain. The sutures must have penetrated the wall of the pouch, as there was a slight discharge from the wound, and later some fluid escaped upon drinking. It is difficult to understand how sutures could be inserted into a tissue of such thinness without penetrating it.

Subsequent History.—The patient left hospital in about three weeks and passed under the care of Dr. Kirby, of Brighton, and although I heard of him from time to time I did not see him again for five years.

He was re-admitted to the Freemasons Hospital in March, 1933, with the following history. Since his operation a tiny sinus had persisted more or less all the time, healing and breaking down from time to time, but giving no trouble in any way. About four months previously other sinuses began to form, he also began to have trouble with the swallowing of solid food, and phlegm formed in his throat on eating.

On examination the skin on the left side of his neck was red, very thickened and indurated, and puckered into folds. In the folds the opening of six sinuses could be seen from which a little pus could be pressed. On probing the sinuses all seemed to converge upon one opening into the œsophagus.

SECOND OPERATION.—An operation was performed, the sinuses were opened up, and most of the thickened skin was cut away after the manner of dealing with an anal fistula with multiple openings. The skin was so suspicious that it was sent to the pathologist for examination.

PATHOLOGICAL REPORT.—Squamous-celled carcinoma was found in the skin and subcutaneous tissue. The growth is in large indurated masses with little keratinization. Mitotic figures are few in number.

On receiving this report radium was inserted into and around the wound and 6200 mgm.-hrs. were given. He developed a good reaction and the wound began to heal and was quite small when he left the hospital.

Two months later Dr. Kirby wrote to me to say that he had been called to the patient one morning and found him gasping for breath. "There was definite stridor, and a swelling at the back of the pharynx; it looked like a retropharyngeal abscess." He was sent into hospital but it was decided not to operate, and he died the same evening.

# *REVIEWS AND NOTICES OF BOOKS*

**The Life of Sir Robert Jones.** By FREDERICK WATSON. Medium 8vo. Pp. 327. Illustrated. 1934. London: Hodder & Stoughton Ltd. 12s. 6d. net.

THIS is a book written with perspicuity, sympathy, and love. The character of Sir Robert Jones is delineated with great exactness: his devotion to his life work, his love of humanity, suffering or otherwise, his geniality, tact, kindness, and sympathy are well shown and without a word too many.

After a brief account of Jones's boyhood days, we are introduced to that dour genius Hugh Owen Thomas, who gave him his first insight into the principles of orthopædic surgery. Then we meet the grown man, whose appointment as Chief Surgeon to the Manchester Ship Canal gave him the opportunity of developing his powers of organization, a training which was to prepare him for the call of the War, where he showed himself a giant and did giant's work.

That Sir Robert Jones was ceaseless in his work for advancing science is shown by the fact that he was the first man in England to make use of the X rays; when in 1895 he heard from a private source of the discovery of the Roentgen rays, he immediately set off for Germany to see for himself the photographic process by which bones could be shown through the body. It is significant of his love for children that the first X-ray taken in England was taken by him, with the help of Dr. Thurstan Holland, to detect the presence of a bullet in a boy's wrist. Indeed, crippled and deformed children were his special care. Unlike Luther, who advised the killing of all deformed infants, his main work in life was curing or alleviating cripples; but specially was he concerned with crippled children.

Perhaps the chief benefit that this great surgeon conferred on the nation was due to his wonderful foresight, during the War, in arranging for orthopædic centres for the men who, he foresaw, would need all the help that surgery could give them when crippled and disabled by wounds and disease. These centres are now established all over England, and many surgeons are at work who were trained by Sir Robert Jones in the special knowledge needed.

The history of the first open-air hospitals for children is of great interest, and the book is well worth reading for that alone (p. 114). There are many delightful stories in the volume, some gay, some sad, some mischievous, and all delectable. The marvellous capacity for hard work, the joy of living, the enjoyment of sport, the genius for friendship, and the fascination which Sir Robert exercised over our American colleagues are fully described, and though to those who did not know him the tale may seem incredible, to those who did it rings true in every line. This account of a life spent in helping others should arouse enthusiasm in every heart.

The book is well produced, and contains 10 illustrations.

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**Intracranial Tumours.** By HARVEY CUSHING, Professor of Surgery, Harvard Medical School. Super royal 8vo. Pp. 150 + xii, with 111 illustrations. 1932. London: Baillière, Tindall & Cox. 26s. net.

THIS volume really consists of a clinico-pathological elaboration of Professor Cushing's series of 2000 verified cases of cerebral tumour. Despite the title of the monograph, it can in no way be regarded as a representative account of intracranial new growths. This is shown by the author's references, which do not include any work carried out by individuals who have not studied at his clinic. The writer's experience is divided into three groups: at Johns Hopkins Hospital, from 1901–12; a Peter Bent Brigham series to 1922; and a third interval from 1922–31. The case and operative mortalities over the period 1901–31 have been compared with those in the three years 1928–31. Thus, over the total period the case mortality has been 20·4 per cent as compared with 13·3 per cent over the period 1928–31, and the operative mortality 13·9 as compared with 9·8 per cent. Operative fatalities comprise all cases of death in hospital following an operation, from any cause whatever, no

matter how long the interval. The average hospital sojourn of the last 100 cases with surgically verified tumours has been thirty-nine days; the author, however, does not specify the average stay in hospital after the actual operation.

Professor Cushing attributes the progress of intracranial surgery over the thirty years to three main factors: the development of a highly specialized surgical technique, more precise localizing diagnoses, and a better understanding of the life history of tumours of different types based on their histogenesis. In regard to the details of improvement in technique, the writer emphasizes seven features: (1) The generally accepted methods of decompression to relieve tension; (2) Such irreproachable wound healing that secondary infections are practically unknown; (3) The separate closure of the galea by buried fine black-silk sutures, which has made the once dreaded fungus cerebri almost forgotten; (4) In place of ether inhalation, the introduction by de Martel of local anæsthesia, now supplemented when necessary by the rectal administration of tribromethanol; (5) The more precise tumour localization which in obscure cases Dandy's ventriculography permits us to make; (6) The use of a motor-driven suction apparatus as an indispensable adjunct to every operation; and (7) The successive improvements in methods of hæmostasis which since 1927 have been most advantageously supplemented by the introduction of electro-surgical devices.

Professor Cushing himself realizes that mere improvement in operative statistics tells us comparatively little, and that what would be more desirable is an account of the remote history, the survival periods, and the working capacity of patients after extirpation of the tumour. As the writer admits, the ultimate case mortality of malignant cerebral gliomata is actually in the region of 100 per cent.

Professor Cushing likens his technical results to prowess at golf. "Games are scarcely worth playing unless one keeps a score. In golf, for example, it is only by the score that a player can tell whether his game is improving or falling off; and only when his competitors keep score by the same standards can it be told by comparison of figures whose game is the poorer." On the same analogy we may regard the writer's score as bogey.

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**The Adrenal Cortex: a Surgical and Pathological Study.** By L. R. BROSTER, O.B.E., M.A., D.M., M.Ch. (Oxon), F.R.C.S., Surgeon to Charing Cross Hospital; and H. W. C. VINES, M.A., M.D. (Camb.), Pathologist, Charing Cross Hospital Institute of Pathology. Demy 8vo. Pp. 94 + vi, with 4 illustrations. 1933. London: H. K. Lewis & Co. Ltd. 6s. net.

IT has long been recognized that some cases of virilism in women are associated with tumours of the adrenal gland; it is probable that their association is that of cause and effect, for removal of the adrenal neoplasm has been promptly followed by the disappearance of the virilism; moreover, in one case at least, the recurrence of the malignant growth was accompanied by the renewed onset of the signs of virilism.

The authors of this monograph start on the assumption that if a female shows the signs of virilism, there must be changes in the adrenal gland, and they show good reason for suggesting that this may be due, not to a neoplasm, but to a hypertrophy of the gland. As such a hypertrophy is unlikely to be obvious on clinical examination, they have opened the abdomen and palpated the adrenal on each side; if one was obviously larger than the other, they have at a second operation removed that one. The results of their operations are recorded in this book, and they are striking though not dramatic. Perhaps one could not expect the extraordinary return to normal that can occur after the removal of an adrenal tumour; there is always the possibility in their cases that both adrenals were hypertrophied, though not equally, or, after the removal of one, the other may take on the morbid change.

The last part of the book is occupied with a discussion of a material found in these adrenals which stains a vivid red with ponceau fuchsin; various reasons are given for considering this an indication of an over-production of a specific secretion.

In the excellent description of the various changes comprised under the heading of virilism we note with surprise one omission; whilst discussing the loss of hair on the head no mention is made of 'apical baldness', though we believe that this is a privilege reserved for the male; in one case on record the diagnosis of an adrenal neoplasm was first suggested by the presence of this limited alopecia.

The book is well written and must arouse great interest amongst surgeons, physicians, and pathologists.

**The Thyroid Gland: Its Chemistry and Physiology.** By CHARLES ROBERT HARINGTON, M.A., Ph.D., F.R.S., Professor of Pathological Chemistry in the University of London. Large 8vo. Pp. 222 + xiv. Illustrated. 1933. London: Oxford University Press. 15s. net.

THE field of thyroid physiology is now so vast that Professor Harington is to be congratulated on the excellent and well-balanced manner in which he has treated the subject. The main theme of the book is to set out concisely yet completely the earlier work on the chemistry of the thyroid gland which led up to his own brilliant researches, and in more detail his own work and that of his collaborators on the chemical constitution and synthesis of thyroxine. On this there is no one better qualified to write, and even for this alone the book is to be thoroughly recommended.

To illustrate the biochemical relationships of the thyroid to the organism as a whole, he has given a reasoned account of the essential physiology and pathology of the gland. The importance of iodine deficiency, whether it be absolute or relative, in the etiology of simple goitre is justifiably emphasized; to bring forward arguments against the etiological significance of iodine is undoubtedly to lack a clear conception of the fundamentals of the subject. Professor Harington in his discussion on Graves' disease points out what pathologists are slowly beginning to realize, that the morphological changes and the biochemical reactions of the thyroid in this condition in no way differ from those of simple goitre, and shares the view of most competent observers that the cause of hyperfunction must be sought outside the gland itself. He forms the logical opinion that there is no good reason for considering the so-called "adenomatous goitre with hyperthyroidism" or "toxic adenoma" and Graves' disease as separate entities. In criticizing the dysthyroidism theory, however, his argument that the thyroid glands from cases of Graves' disease are inactive when administered to animals is unconvincing, for in the same way as we know that the gland pours out its active secretion at an increased rate so that none is retained, so the hypothetical "toxic" secretion would be poured out and not be retained. Moreover, many clinicians would not agree with his statement that the beneficial effect of iodine is but of short duration. The argument, however, which Professor Harington brings forward, that no derivative of thyroxine containing less than the full complement of iodine exhibits any toxic properties whatever, definitely refutes the hypothesis.

Professor Harington's book should be read by all those who are interested in the thyroid gland. There is a good bibliography.

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**The Study of Anatomy written for the Medical Student.** By S. E. WHITNALL, M.A., M.D., B.Ch. (Oxon), M.R.C.S., L.R.C.P., F.R.S. (Canada), Robert Reford Professor of Anatomy, McGill University, Canada. Second edition. Crown 8vo. Pp. 93 + viii. 1933. London: Edward Arnold & Co. 4s. net.

PROFESSOR WHITNALL, the author of this book of wisdom, is distinguished both as a writer and as an anatomist. He has generously put his reading and experience at the service of the medical student. If the student were to accept the advice offered and practise the precepts of the author, then he would be wise in his adolescence instead of only in his old age.

The author opens with a proper definition of anatomy and at once proceeds to make clear what the subject is. His insistence that topographers, mappers, sketchers, and markers of the body shall consider what the structures are for and how they work is welcome. An eloquent passage on the skin illustrates the author's attitude, and we quote its final sentences: "It [the skin] is indeed the finest fighting tissue. But it does surprisingly more than serve as a protective covering against such manifold influences, for it will also act as a regulator of temperature, an excretory organ, and the largest and most versatile of our sense organs."

The second chapter of the book is concerned with practical methods of study—sharpening scapels, describing directly the dissected structure, drawing it, looking at other dissections, seeing the same region from some other approach or in cross-section, etc.

The author continues to act as Nestor in chapters on books, lectures, and examinations; to rub in common sense and plead for reflection and thinking as substitutes for memorizing—a drudgery that seems worse in medicine than in any other study. Point is given to common-sense remarks by a skill in apt quotation. "It is an idle and pernicious habit to ask for information on any question before bringing one's own judgment to bear on it" (Bridges).

The last chapter opens rather depressingly, for it begins with what one ought to read: then out come the usual first-past-the-post best hundred classics. Then the author, having put his penny in the cultural plate, proceeds in the raciest manner to string together the titles of his real bedside friends: *King Solomon's Mines, Treasure Island, Robbery under Arms,* and all the others.

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**Operating Room Procedure for Nurses and Internes.** By HENRY C. FALK, M.D., F.A.C.S., Clinical Professor of Gynæcology, New York University, and Bellevue Hospital Medical College, etc. With Foreword by EUGENE H. POOL, M.D., New York. Second edition. Large 8vo. Pp. 413 + xxxii, with 328 illustrations. 1934. New York and London: G. P. Putnam & Sons. $3.

THIS is certainly one of the most practical books on the subject which we have yet seen. It begins with a description of the principles and details of the operating room and then discusses the duties assigned to the various grades of nursing sisters, the design and making of the dress, etc., to be provided for surgeons and nurses, the provision of the operating theatre stores, and the best method of keeping them. The chapter on hand preparation is short, but the methods described are far more complicated than those used in this country. In order to preserve silence in the theatre, a manual signal system has been invented (which we confess we find it difficult to take seriously); thus by holding up the hand with one or more fingers turned in various directions the nurse is instructed to fetch some particular instrument or dressing. The larger second portion of the book consists of a description of various operations, chiefly those concerning the abdomen, pelvis, and female genital organs. The clearness of the line drawings is a commendable feature of the book.

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**La Sténose hypertrophique du Pylore chez le Nourisson.** By J. POUCEL, Chirurgien des Hôpitaux de Marseille. 7¾ × 5 in. Pp. 108 + viii, with 16 illustrations and 8 plates. 1934. Paris: Masson et Cie. Fr. 20.

THIS little manual gives a very complete study of the clinical and pathological aspects of the disease. In stressing the necessity for early diagnosis followed by surgical intervention the author reminds us that Fredet practised and published his account of the modern operation some years before Rammstedt, whose name is commonly associated with the procedure. In diagnosis the author favours repeated short radiographic examinations, which he regards as harmless and essential. In this he would appear to differ from many English pædiatricians who reserve the X-rays for the occasional doubtful case rather than for routine use, basing this practice on their experience that diagnosis in skilled hands is extraordinarily accurate, whilst the barium meal is not altogether harmless. We agree with the author that many lives are still being lost by a failure to recognize the condition in its earlier stages when its surgical treatment is safe and the results of operative intervention are most satisfactory.

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**The Enlarged Prostate and Prostatic Obstruction.** By KENNETH M. WALKER, F.R.C.S., M.A., M.B., B.C., Lecturer in Venereal Diseases, St. Bartholomew's Hospital, etc. Second edition. Demy 8vo. Pp. 223 + xiv, with 63 illustrations. 1933. London: Oxford University Press. 14s. net.

IN his preface to this edition the author explains the alteration of the title of this book (from *The Enlarged Prostate* to *The Enlarged Prostate and Prostatic Obstruction*) by the wider recognition of the part played by minor changes in the neck of the bladder and by the improvement in the instruments used for per-urethral resection; he points out that he is dealing with prostatic obstruction rather than with prostatic enlargement.

The reader will find an excellent account of the anatomy and function of the prostate, with some interesting remarks on the rôle played by the trigonal muscle in the act of micturition, and, in the third chapter, a very complete and well-balanced summary of the various theories on the pathology of prostatic obstruction.

We naturally look to the chapter on per-urethral prostatectomy as being of most interest to the modern surgeon; there we find a clear account of the operative technique and of the special instruments that have to be employed. Of even more importance at the present time are the author's remarks on the indications for this operation; he considers that the

key to success lies in the careful selection of cases and that the per-urethral approach is not to be looked on as a substitute for prostatectomy. He would employ this method for cases in which the intravesical portion of the prostate is affected whilst the total volume, as estimated by bimanual examination, is but little increased; and again for cases in which total prostatectomy is contra-indicated because the patient's general condition is too poor or when he is suffering from cardiac, pulmonary, or kidney lesions. In these conditions the author considers it an excellent alternative to catheter life or a permanent suprapubic drainage, and the fact that the obstruction may recur is not the drawback one might suppose, for the inconvenience is so slight that a patient will return for further treatment.

Not the least valuable part of this book are the references at the end of each chapter; they are well chosen and not too numerous.

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**Trattato di Patologia chirurgica generale e speciale.** By Professor OTTORINO UFFREDUZZI (Turin). Large 8vo. Vol. II. Pp. 1199 + vii, with 25 plates and 448 illustrations. Turin: Unione Tipografico-editrice Torinese. 1934. L. 145.

IN reviewing the first volume of this admirable work (BRIT. JOUR. SURG., 1933, xxi, 165) it was remarked that no acknowledgement was made of the plates taken from this journal's ATLAS OF PATHOLOGICAL ANATOMY. The omission has now been made good.

That one man should be able to cover the whole field of general surgery to-day is a notable tribute to his energy, his memory, and his catholicity of interest. That he should lend to twelve hundred pages an air of all having been written to-day is even more notable. This volume deals with the capillaries, veins, and lymphatics; the central and peripheral nervous system; the skin and its appendages; the respiratory and digestive systems; and the urogenital system. The book differs from, and is superior to, a great many large continental text-books, in that though a vast deal of current literature is traversed, it is all presented through the medium of the author's own mind, illustrates his practice, and here and there discloses his little prejudices. In every instance the general discussion of the physiology and pathology of a given system offers the student a reasoned and reasonable basis for diagnosis and treatment.

Only one or two points can be noticed. There is an account of successful experiments on the regeneration of the sympathetic after division, which is used as an argument in favour of ganglionectomy. In dealing with tumours of the lung, the accounts of lobectomy hardly reflect present-day success. The parathyroids have a good deal of consideration, but there are more specific results than are here indicated. In speaking of the physiology of the stomach the work of Bayliss and Starling, though perhaps implied, is hardly given due weight. Without, however, reading every word of the text attentively, it would not be fair to allege neglect of any piece of recent work, since again and again a casual sentence occurring outside the general discussions indicates a familiarity with contributions to the literature not specifically named amongst the ample acknowledgements.

An achievement of which the author and his University may be proud, the book deserves translation into English. It would be a colossal task; probably it will in fact be transcribed into American, and thus students here will have the benefit.

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**The 1932 Year Book of Radiology.** Edited by CHARLES A. WATERS, M.D., Associate in Roentgenology, Johns Hopkins University, etc. (Diagnosis); and IRA I. KAPLAN, B.Sc., M.D., Director, Division of Cancer, Department of Hospitals, New York, etc. (Therapeutics). Pp. 750, with 495 illustrations. Chicago: The Year Book Publishers, Inc. $6.00.

THIS book contains abstracts of the most important articles which have appeared in the radiological journals of Europe and America during the past year. The abstracts are concise and clear, and a reference to the original article is given with each. The illustrations are numerous and maintain a high standard. There is an index both of subjects and authors at the end, so the book is ideal for reference purposes, while in many cases the abstracts are so clear that it will not be necessary to refer to the original, especially as these may be in German, while the abstracts are all in English. The book incorporates and summarizes all the recent advances in radiology and radium therapy during the past year, so that it should have a wide appeal to physicians and surgeons.

**United States Army X-ray Manual.** Authorized by the Surgeon-General of the Army. Second edition, rewritten and edited by Lieut.-Colonel H. C. PILLSBURY, M.D., U.S.A. Crown 8vo. Pp. 482 + xvii, with 228 illustrations. 1932. London: H. K. Lewis & Co. Ltd. 25s. net.

WHEN the first edition of this work was published, it was found to be not only an excellent guide to war radiology, but also a very good elementary work on radiography in general. In revising the work the present writer has had the latter object more in mind than the former. The chapter on localization has been cut down from ninety-three pages to twenty-nine, and much that was useful has been omitted. For instance, the method of parallex which was extensively used in our Casualty Clearing Stations in the world war, as one of the quickest and most accurate, is not described. Surely the localization of foreign bodies must be taught to an army surgeon as long as rifles, machine-guns, shells, and bombs are issued to the rest of the army. When the use of these weapons discontinues, the need for an army X-ray manual will cease.

The war portion of the book has suffered little revision: for instance, the field unit described and illustrated is apparently the same as in the previous edition and is that used in the Great War fifteen years before. We civilians who worked in the British Army in the war found our commanders terribly conservative, but we trust they will in the next war be a little more abreast of the times than this. The need for an immediate and urgent report in cases of gas infection is not mentioned—a most important thing in a Base Hospital or Evacuating Station.

In the present edition an attempt has been made to separate technique into a distinct section, but the technique of some sections still remains in the text, e.g., in the chapter on sinuses. One of the features of the first edition was the excellent description and photographs of the various techniques employed in different parts of the body. These have now practically disappeared. Many illustrations have been added to the various sections, and some of them bear evidence of considerable revision, notably that on the thoracic viscera. Some sections are hardly brought up to date. We are glad to see that the chapter on therapy has been omitted: in the original edition it was very bad.

Taking the work as a whole, therefore, it is to be regarded less as an army X-ray manual than as an elementary work on general radiography. Much that was valuable in the first edition has disappeared, and, though the work has been brought up to date, it is doubtful if the revision has proceeded along the best lines for the instruction of an army radiologist.

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**Radiologic Maxims.** By HAROLD SWANBERG, B.Sc., M.D., F.A.C.P., Editor of *The Radiological Review*. With a Foreword by HENRY SCHMITZ, A.M., M.D., LL.D., F.A.C.P., F.A.C.S. Large 8vo. Pp. 127. 1932. Quincy, Ill.: Radiological Review Publishing Co. $1.50 net.

THIS little book is made up of maxims on radiology and radium therapy, with short abstracts from the literature—or what one might call "famous sayings of the great". Some of the maxims are good and some indifferent, while many are platitudes, such as (p. 39), "Roentgen examinations are indispensable to the urologist." Generalizations are made which by themselves are not entirely true, such as (p. 102), "The therapeutic effects of radium and radon are the same". It is difficult to see who is going to be instructed by these. Some of the statements are of interest as illustrating the views of the author or authorities quoted—for example (p. 36), "Duodenal ulcer is about ten times as frequent as gastric ulcer", but even here the facts convey little unless amplified.

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**Radium: Production, General Properties, Therapeutic Applications, Apparatus.** Demy 8vo. Pp. 346, illustrated. 1933. London: Chemical Service Co. Ltd., Sunic House, Kingsway, W.C.2.

THIS is not a book written specially for medical men, though there are many points in it which are useful for doctors. It has been produced for the purpose of dealing briefly with the fundamental principles of radium therapy and of the precautions to be observed by the radium therapeutist. There are many points in it which will be found useful to those entering upon the study of radium therapy.

**Radium and Cancer.** By H. S. SOUTTAR, C.B.E., M.D., M.Ch. (Oxon), F.R.C.S., Surgeon to the London Hospital, etc., Pocket Monographs on Practical Medicine. F'cap 8vo. Pp. 64 + viii, with diagrams. 1932. London: John Bale, Sons & Danielsson, Ltd. 2s. 6d. net.

THIS is a little book of sixty-four pages only and costing 2s. 6d.; yet it includes some of the most important facts relating to the treatment of malignant disease by radium. To anybody who is commencing the study of radium treatment of cancer this book will repay perusal. It describes quite briefly the physics of radium, the apparatus and means of application, the sensitivity of the tissues to radium, and details of the application of radium in various parts of the body. In addition it sets out in a final chapter a warning note against the accidents which too often have followed its indiscriminate use.

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**A Short History of Some Common Diseases.** By Divers Authors. Edited by W. R. BETT. Demy 8vo. Pp. 211. London: Humphrey Milford (Oxford University Press). 10s. 6d. net.

THE plan of this book is indicated by the title, although this gives no clue to the amount of information and interest which have been crowded into its pages.

It may be at once said that the dedication hides yet another illusion about the editor which may only be known to few, for it is a fact that Mr. W. R. Bett who has assembled this remarkable collection of essays is not the old and satiated person that the dedication suggests, but one who ought to have many years of usefulness during which, let us hope, he will still further encourage the study of medical history, not as a detached subject but as a prelude to the knowledge and understanding of disease.

This little volume contains no fewer than sixteen articles, each one complete in itself, and dealing with most of the subjects under which morbid conditions are usually considered, starting with Acute Infectious Disease and finishing with Malingering. It includes subjects like Venereal Disease, Rickets, Endocrine Disorders, Arthritis, Tonsils and Adenoids, and Malignant Disease, as well as others.

The names of the authors are a guarantee of the completeness with which the subjects are dealt. Sir Humphry Rolleston, Sir John Broadbent, Sir D'Arcy Power, E. M. Brockbank, and John D. Comrie are all sufficiently well known as medical historians to command respect. Some of the chapters by less well known historians make delightful reading also, and the story of Tuberculosis by Professor John Fraser of Edinburgh, and that of Appendicitis by W. R. Bett, are especially worthy of mention.

In several of the articles there are quotations from older works and some of them give lists of references, but the way in which nearly every article unmasks the personality of its author makes the book the more delightful and valuable. How nice it would be if some future edition could be embellished by a few portraits!

This work is not only likely to attact the serious student, but will serve as an introduction to the history of the Healing Art, and it might well be expected that those preparing themselves for the higher reaches of the profession should be acquainted with its pages.

There are useful indexes both of subjects and names.

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**Lessons on the Surgical Diseases of Childhood.** By WILLIAM RANKIN, M.B., Ch.B. Royal 8vo. Pp. 190. Illustrated. 1934. Glasgow: Alex. Macdougall. 21s. net.

DR. RANKIN tells us in his preface that the book is a summary of the lecture and demonstration notes which he gives to first-year students. He states that he intends it to be no more than a groundwork of introduction to the subject, and this is a correct description, for, while he covers a wide field in a superficial review, there is insufficient detail to make the book of value to the inquiring reader. For example, Chapter XI, which purports to deal with the common abdominal conditions of childhood, is condensed within a space of some thirteen pages including illustrations. It is obvious, therefore, that the volume is something in the nature of a catalogue of the surgical affections of childhood, elaborated at certain points in relation to detail which makes a special appeal to the author.

The volume is divided into twenty-six sections, and of these the best is undoubtedly the first, dealing with hare-lip and other developmental lesions of the face. It is obvious

that this is a section in which Dr. Rankin is particularly interested, and if the other sections had been brought to a similar standard the result would have afforded more interesting and satisfying reading than is the case under existing conditions. There are many points open to criticism, but these are too numerous to call for individual notice; it may, however, be permitted to refer to one or two. It is probably unwise to suggest that "only very rarely should the premaxilla be removed entirely." This would seem to presuppose that an incomplete removal may be adopted with relative frequency, but would it not be more correct to recommend that under no circumstances should the premaxilla be removed? This recommendation has the entire support of modern opinion. Again, in describing the operative treatment of empyema the statement is made, "the removal of two inches of the second rib beneath the angle of the scapula when the arm is laid against the chest wall is preferred to removal of a piece of rib in the mid-axillary line." It is probable that this statement is the result of imperfect wording, but as it reads it would seem to suggest that a portion of the second rib should be removed. Presumably the author intends to recommend removal of a rib at a lower level than the scapular angle, i.e., the 9th or 10th rib, but as it stands the statement is apt to create an entirely wrong impression in the student mind. We question, too, whether it is judicious to state that "in empyema the old operation of free drainage through a fairly large drainage tube has not been replaced by any of the more modern methods." It is a pity that no attempt has been made to distinguish between the kind of treatment adopted in the pneumococcal and in the streptococcal types of the disease.

Dr. Rankin, in speaking of the operation for mastoid infection, says that once one is familiar with the small area involved and the danger spots, the operation is easy, and can be performed with a small Volkmann spoon. This is surely an optimistic view to adopt, and we question if it would have the approval and the blessing of those who are interested in operations of this class.

We are in strong disagreement with some of the author's views on embryology and comparative anatomy. It is surely disrespectful, to say the least of it, to speak of the teratoma as a 'blasted ovum', and there is no evidence that webbed fingers constitute a throw-back to the flipper limb of an amphibian ancestor. We disagree, too, with the statement that the development of the spinal cord is an antenatal operation necessitating the making and the healing of a huge wound. This statement may have certain attractions in the way of descriptive writing, but it is scarcely an accurate presentation of the facts.

The volume is profusely illustrated, though a certain number of the photographs are inserted without descriptive legends. The value of the book would be increased by the addition of an index. The printing of the text and the presentation of the book from the publisher's point of view leave nothing to be desired.

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**Bone Growth in Health and Disease.** By H. A. HARRIS, D.Sc., M.B., B.S. (Lond.), M.R.C.S., M.R.C.P., Professor of Clinical Anatomy, University College and University College Hospital, London, etc. Crown 4to. Pp. 248 + xviii, with 201 illustrations. 1933. London: Humphrey Milford (Oxford University Press). 34s. net.

IN this interesting and discursive monograph, Professor Harris elaborates views on bone growth which he has already published elsewhere.

The first part deals with the lines of arrested growth in the long bones of children which form during a period of starvation, infection, or disease.

The second part is concerned with bone growth in deficiency diseases. The problem is approached from two aspects: (1) The general principles of ossification in the skeleton; and (2) A detailed consideration of the changes seen in proliferating cartilage, calcifying cartilage, and bone in terms of histological, radiographic, and morphological criteria. Emphasis is laid on the differences of nutrition in these three processes, and it is suggested that each is controlled or influenced by a different vitamin, and by other substances of a somewhat speculative nature.

The origin, function, and fate of the osteoblast, and of the osteoclast, are still debatable problems. The author believes that the osteoblast is an altered cartilage cell possessing specific bone-forming properties, but presents no new or convincing evidence in support of this traditional view. It would have been instructive if he had discussed in critical fashion the views of Leriche and Policard, and Greig, that bone formation is merely a metaplasia of

fibrous connective tissue in which calcium and phosphorus are deposited, and that the osteoblast is simply an altered fibroblast with little, if any, bone-forming property.

Part III is devoted to a study of the significance of the fundamental processes of growth and repair in skeletal disease. This section is stimulating and provocative. From observations of the cells of normal human cartilage it is claimed that the dividing cells are arranged in a 'mitotic annulus' at the end of a bone, and from this limited area growth occurs. The author believes that synovial fluid is produced by the disruption of the cells of articular cartilage and is absorbed by the synovial membrane.

Harris has carried the researches of Robinson and Fell on the ferment phosphatase to a further stage, and has demonstrated the gradual accumulation of glycogen in the cartilage cell as the cell ages, to disappear entirely when cartilage is replaced by bone. He suggests that the glycogen is hydrolysed to hexose phosphoric esters which under the action of phosphatase and the calcium of the circulating body fluids leads to the deposition of an insoluble phosphate of calcium in the matrix. How in the absence of a blood-supply calcium reaches the cartilage cell is a problem that requires further research, but it seems certain that the explanation of calcium deposition is a biochemical, and not a mechanical one.

Although this book is fragmentary in its presentation of the subject of bone growth, it makes attractive reading, and should give fresh stimulus to research on a problem which is still to be solved.

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**The Spread of Tumours in the Human Body.** By RUPERT A. WILLIS. M.D., B.S., D.Sc. (Melbourne), Pathologist to the Alfred Hospital and to the Austin Hospital for Chronic Diseases, Melbourne. Demy 8vo. Pp. 540 + x, with 103 illustrations. 1934 London: J. & A. Churchill. 25s. net.

IT is unusual and most refreshing to find a pathologist, or for that matter any other medical author, attacking a subject from an original point of view and departing completely from the scissors-and-paste methods so common and so tedious in medical literature. Dr. Willis believes that a close study of the modes of spread of tumours may help to teach us much of their nature and origin, and his book, the outcome of many years of careful study of his own autopsy material and of exhausting sifting of an immense volume of literature—his bibliography fills 65 pages and contains more than 2000 references—is an ample justification of his claims.

The book is divided into two parts. In Part I the general problem is discussed, and excellent chapters appear on the routes of dissemination, the reaction of stroma, and the comparative susceptibilities of different tissues to tumour growth, etc.; while Part II deals with secondary neoplastic disease of individual organs and tissues.

The book is full of valuable information and carefully weighed criticism of existing theories, criticisms which are inspired by a rare soundness of judgement. It widely exceeds the limits suggested in the title, for it is a general exposition of tumour formation, and those who delight in theory will be disappointed, for the writer deals with facts and discusses theories mainly to explode them. Dr. Willis apologizes in his preface for appearing iconoclastic, but his outspoken criticisms are one of the most attractive features of the book, and he has done a great service in exposing the fallacies of much teaching that has become traditional.

In a book which is in every respect so excellent it is difficult to select parts for special commendation. It will appeal to all lovers of truth, and to the morbid anatomist and surgeon alike its value cannot be exaggerated.

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**Spinal Anesthesia: Technic and Clinical Application.** By GEORGE RUDOLPH VEHRS, M.D., Salem, Oregon. Large 8vo. Pp. 269, with 81 illustrations. 1934. London: Henry Kimpton. 21s. net.

ANY reader unacquainted with the practical side of spinal anæsthesia reading this book might excusably make the deduction that the question of anæsthesia for any operation in surgery had been solved so perfectly that it would be scarcely worth while to make any further researches on this subject. From which it is clear that the author is an enthusiast for the method. He seeks to convince us that, by the adoption of his technique, the anæsthesia is absolutely controllable. The sequence of events is fixed by what he is pleased to call "unalterable laws of spinal anesthesia." He starts out with the assumption that novocain introduced

into the spinal canal diffuses only by the influence of gravity. He relies upon fixation of the novocain locally to reduce its concentration to a value harmless by the time it has risen to the cervical region. He advocates keeping the patient exactly horizontal on his side for some minutes after injection to obtain this fixation before adopting the Trendelenburg position to cause upward spread. There is nothing new in his technique but this.

An interesting chapter is devoted to regional spinal anæsthesia. By this is meant sensory without motor block, which according to the author it is possible to obtain at will by reducing the dose of the drug, because a higher concentration is needed for motor than for sensory block.

There are a number of experiments described in which the drug was injected into the cisterna magna of dogs which prove that if the concentration is high enough death will follow. In view of Koster's statements it is well to have this fact, which others have demonstrated, insisted upon.

The book does not add very much to our knowledge of this interesting subject.

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**Verletzungen und Krankheiter der Kiefer.** By GEORGE PERTHES (Tübingen) and EDUARD BORCHERS (Aachen). Royal 8vo. Pp. 623 + xiv, with 234 illustrations. 1932. Stuttgart: Ferdinand Enke. Paper covers, RM. 75; bound, RM. 78.

THE first edition of this book was published twenty-five years ago by Perthes. The second edition has been prepared by the pupil of Perthes—Eduard Borchers. The work comprises a complete treatise on affections of the jaws together with a very extensive bibliography. References are made to the English and French literature on the subject, and the arrangement of the book is effective. Deformities are dealt with fully, due regard being paid to congenital anomalies. Acquired deformities are discussed in acromegaly, cretinism, idiocy, thymus affections, and rickets. The treatment of fractures of the jaws is given fully, and the importance of the help of a dental surgeon is stressed. Dislocation of the mandible, diseases of the temporo-mandibular joint, and inflammation in all its varieties are exhaustively surveyed. The section devoted to neoplasms is particularly good. The question of the classification of odontomata is discussed, and a simple classification put forward. Regarding the treatment of sarcomata, we note the advisability of radical operation instead of spending time using radiological methods, which are not followed by any striking improvement. The author finds carcinoma more common than sarcoma affecting the jaws, and recommends radical removal where possible.

The book is well worth study. The print is clear and the illustrations, histological, naked-eye, and radiological, are good.

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**Gelenksteifen und Gelenkplastik.** By Professor Dr. ERWIN PAYR (Leipzig). Vol. I. 11 × 8 in. Pp. 880 + xiv, with 240 illustrations. 1934. Berlin: Julius Springer. Paper covers, RM. 120; bound, RM. 124.8.

IT is difficult to do justice to this magnificent monograph in the space at our disposal. For more than thirty years it has been in preparation. The author had at first planned a book dealing with the technique of plastic joint reconstruction; but he has been so greatly impressed with the need for laying a good foundation of biology, pathology, anatomy, and biochemistry that at present he has had to be content with the presentation of these aspects of the problem with which are associated a rich fund of clinical material and experimental evidence. A further volume is promised which will deal with the technique of operative procedures.

The opening chapters deal with the history of the subject, the development of joints, comparative anatomy, and composite structure of joints in animals and man. Then follows a description of the origin, nature, and causes of different types of ankylosis in constitutional diseases, infections, and injuries. The pathological anatomy and physiology of ankylosed joints are described in great detail and then the phenomena of the formation of false joints. After this natural formation of false joints there follows a discussion of the experimental attempts at joint regeneration. This section concludes with a most instructive summary of the application of the lessons of animal experiments to the human problem. The most significant point is that in animals the rapid regeneration of a joint largely depends upon its early active use, and it is argued that this principle should be applied in human surgery.

The clinical aspects of ankylosis are analysed with great care. Kinetic factors, psychology, pain, and tonus are all discussed. The fibrous and bony types of ankylosis, the effect of

ankylosis on the rest of the body, and the compensatory changes which take place are considered in connection with each individual joint. A consideration of the various causative factors in ankylosis, congenital, inflammatory, infectious, constitutional, and endocrine disturbance, is followed by that of the technical methods of determining its nature and cause. This volume of the work concludes by a lengthy discussion of prognosis and the indications or contra-indications for plastic operations on ankylosed joints.

The work is of a thorough and monumental character, it is well printed and beautifully illustrated, and not only the many pupils, past and present, of its distinguished author, but the whole surgical world is richer for its appearance.

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**Synopsis of Surgery.** By ERNEST W. HEY GROVES, M.S., M.D., B.Sc. (Lond.), F.R.C.S., Consulting Surgeon to the Bristol General Hospital, etc. Tenth edition. Crown 8vo. Pp. 693 + viii, with 163 illustrations. 1933. Bristol: John Wright & Sons Ltd, 17s. 6d. net.

IN the latest edition of this well-known book the whole text has been carefully revised and brought up to date. The sections on the radium treatment of malignant disease, the surgery of the sympathetic nervous system, and the vaseline pack or Winnett-Orr method of treating septic bone conditions have been re-written, and a new chapter has been added giving an excellent outline of the principles underlying and the features of the more important amputations. The fact that this is the tenth edition of the book that has appeared since its original publication in 1908 is sufficient guarantee of its continued usefulness to the student and of its popularity.

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**Il Cancro del Retto.** By VITTORIO PETTINARI. Crown 4to. Pp. 225 + iv, with 121 illustrations. 1934. Bologna: Licinio Cappelli. L. 30.

THIS monograph, written by one of Professor Castiglioni's staff in the Institute of Surgical Pathology in the Royal University of Milan, is based on an extensive material and upon wide study of the literature.

It deals systematically with every aspect of the subject, and is fully illustrated, most of the pictures being adequate. It cannot be said that anything very new is incorporated, either in the pathogeny, diagnosis, or treatment, and for students there is rather too much record of numerous other surgeons' opinions, without clear indication of what the author himself thinks. He has, he says, frequently observed the initial stages of cancerization, and considers that differences are recognizable between the changes occurring when it commences in normal tubules and adenomata respectively. The symptomatology, clinical forms, and diagnosis present nothing exceptional, though it is noteworthy that the proliferative type seems more frequent in Italy than the ulcerative. Statistics are collected from all sources. The operative measures recommended do not commend themselves. Colostomy is used only when obstruction actually exists, and the lower end of the colon, after perineal operation, is abandoned in the depths of the wound, which is allowed to granulate under Carrel-Dakin dressings. There is a good bibliography.

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**Die Embolie.** By Dr. SIGURD FREY (Königsberg). Royal 8vo. Pp. 178, with 36 illustrations. 1933. Leipzig: Georg Thieme. RM. 12.

THIS is a monograph in German by Dr. Frey, the Privatdozent for Surgery in the University of Königsberg. It is a description of the varieties of emboli, their causes, and their effects.

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**Beitrage zur Mund und Kieferchirurgie.** By Prof. Dr. G. AXHAUSEN (Berlin). Part 2 of *Deutsche Zahnheilkunde*, edited by OTTO WALKHOFF (Berlin). Large 8vo. Pp. 117, with 128 illustrations. Leipzig: Georg Thieme. M. 12.60.

A USEFUL contribution to the surgery of the mouth and jaws has been made by Axhausen. A work of this size is not intended to cover the whole field; the main aspects of the subject have been dealt with. Numerous clinical histories are given to illustrate various facts. The pictures are very good. The subject of plastic surgery of the face has been introduced. Profit will result from a perusal of this book.

**Chirurgie de la Main. Plaies, Infections, Chirurgie réparative.** By Dr. MARC ISELIN, Ancien Interne des Hôpitaux. Royal 8vo. Pp. 339 + xii, with 111 illustrations. 1933. Paris: Masson et Cie. Fr. 55.

THIS monograph deals with wounds, infections, and the reparative surgery of the hand. In it the author has incorporated the work of Kanavel, Bunnell, and others. From an extensive personal experience at the St. Louis Hospital, Paris, he advocates some useful technical modifications, e.g., in regard to skin incisions for drainage, the site for drainage of tendon-sheaths, and so on; but on the whole he conforms with modern surgical practice. The book is an excellent work of reference.

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**Kleine Chirurgie.** By HANS KURTZAHN (Königsberg i. Pr.). Second edition. Large 8vo. Pp. 462 + viii, with 167 illustrations. 1932. Berlin and Vienna: Urban and Schwarzenberg. Paper covers, RM. 13.50; bound, RM. 15.

WE are impressed with the practical value of this book. The methods advocated are represented pictorially. The chapter on bandaging we think might have been more comprehensive. Anæsthesia in all its forms is discussed fully. The various types of dental forceps are illustrated, and the methods of teeth extraction are given. The book can be recommended as a short surgical work.

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**Allgemeine und spezielle chirurgische Operationslehre.** By Dr. MARTIN KIRSCHNER (Tübingen). Vol. V/1. Die operative Beseitigung der Bauchbrüche (M. KIRSCHNER). Die Eingriffe an den weiblichen Geschlechtsorganen (G. A. WAGNER). Royal 8vo. Pp. 395 + x, with 304 illustrations. 1933. Berlin: Julius Springer. Paper covers, RM. 78; bound, RM. 86.

THIS is an operative surgery book dealing with hernia of various descriptions, and with gynæcological operations. It is well illustrated with a large number of coloured plates, but has nothing particular to recommend it in preference to similar publications in the English language.

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## *BOOK NOTICES*

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.*]

**The Bristol Medico-Chirurgical Journal: Combined Index for Volumes 1–50 (1883–1933).** Medium 8vo. Pp. 72. 1934. Bristol: J. W. Arrowsmith Ltd. 3s.

**Anatomy of Animal Types: for Students of Zoology.** By E. A. BRIGGS., D.Sc., Assistant Professor of Zoology, University of Sydney. Demy 8vo. Pp. 250 + xxii. 1934. Sydney: Angus & Robertson Ltd. London: Australian Book Co. 10s. 6d. net.

**Acute Intestinal Obstruction.** By MONROE A. MCIVER, M.D., Surgeon-in-Chief, Mary Imogene Bassett Hospital, Cooperstown, N.Y. $10\frac{1}{2} \times 7$ in. Pp. 430 + xviii, with 62 illustrations. 1934. New York: Paul B. Hoeber Inc. $7.50.

**The Clinical Management of Horseshoe Kidney.** By ROBERT GUTIERREZ, A.B., M.D., F.A.C.S., Chief of Clinic of the Department of Urology, James Buchanan Brady Foundation of the New York Hospital, etc. With a Foreword by Dr. EDMOND PAPIN (Paris). $10\frac{1}{2} \times 7\frac{1}{2}$ in. Pp. 143 + xxiv, with 52 illustrations. 1934. New York: Paul B. Hoeber Inc. $3.00.

**La Pratique chirurgicale illustrée.** By VICTOR PAUCHET. Fasc. XIX. Super royal 8vo. Pp. 295, with 249 illustrations. 1934. Paris: G. Doin et Cie. Fr. 70.

**Benign Tumours in the Third Ventricle of the Brain: Diagnosis and Treatment.** By WALTER E. DANDY, Adjunct Professor of Surgery, the Johns Hopkins University. $10 \times 6\frac{1}{2}$ in. Pp. 171 + viii, with 120 illustrations. 1934. London: Baillière, Tindall & Cox. 22s. 6d.

**Bulletin der Schweiz. Vereinigung für Krebsbekämpfung.** Herausgegeben unter Mitarbeit des Eidg. Gesundheitsamtes. February, 1934. 1st year, Fasc. 2. Large 8vo. Pp. 81–144, with 30 illustrations. 1934. Bern: Hans Huber. No price given.

**The Story of the Royal Infirmary, Sunderland.** By WILLIAM ROBINSON, M.S., M.D. (Dunelm.), F.R.C.S. Large 8vo. Pp. 135 + vi, with 10 plates. 1934. Sunderland: Hills & Co. (Sunderland) Ltd. Cloth, 4s.; paper covers, 3s.

**Lord Lister: the Discoverer of Antiseptic Surgery.** By C. J. S. THOMPSON, M.B.E., Hon. Curator of the Historical Collection of the Museum of the Royal College of Surgeons of England. Crown 8vo. Pp. 99. Illustrated. 1934. London: John Bale, Sons & Danielsson, Ltd. 5s. net.

**Die parasagittalen Meningiome.** By Dr. H. OLIVECRONA (Stockholm). $10\frac{3}{4} \times 7\frac{3}{4}$ in. Pp. 144, with 145 illustrations. 1934. Leipzig: Georg Thieme. Paper covers, M. 24; bound, M. 26.

**Die pathologisch-anatomischen Grundlagen der Chirurgie des Rektumkarzinoms.** By Priv.-Doz. Dr. HEINRICH WESTHUES (Erlangen). With a Foreword by Prof. Dr. SCHMIEDEN (Frankfurt a. M.). $11\frac{3}{4} \times 8$ in. Pp. 113 + xii, with 107 illustrations. 1934. Leipzig: Georg Thieme. Paper covers, M. 29.50; bound, M. 32.

**Narkose zu operativen Zwecken.** By Dr. HANS KILLIAN (Freiburg i. Br.). Super royal 8vo. Pp. 406 + viii, with 165 illustrations. 1934. Berlin: Julius Springer. Paper covers, RM. 24; bound, RM. 26.80.

**Die Wirbelgelenke.** By Priv.-Doz. Dr. MAX LANGE (Münich). $10 \times 6\frac{1}{2}$ in. Pp. 121 + vi, with 59 illustrations. 1934. Stuttgart: Ferdinand Enke. RM. 7.

**X-ray and Radium Injuries: Prevention and Treatment.** By HECTOR A. COLWELL, M.B., Ph.D., M.R.C.P., D.P.H.; and SIDNEY RUSS, C.B.E., D.Sc., F. Inst. P., the Barnato Joel Laboratories, Middlesex Hospital. Demy 8vo. Pp. 212 + xii. Illustrated. 1934. London: Oxford University Press. 14s. net.

**The Management of Fractures, Dislocations, and Sprains.** By JOHN ALBERT KEY, B.S., M.D., Clinical Professor of Orthopedic Surgery, Washington University School of Medicine; and H. EARLE CONWELL, M.D., F.A.C.S., Orthopedic Surgeon for the Tennessee Coal, Iron and Railroad Company, Birmingham, Ala. Imperial 8vo. Pp. 1164, with 1165 illustrations. London: Henry Kimpton. 63s. net.

**The Origin of Cancer.** By J. P. LOCKHART-MUMMERY, M.A., M.B., B.C. (Cantab.), F.R.C.S., Senior Surgeon, St. Mark's Hospital, etc. Large post 8vo. Pp. 150 + x, with 29 illustrations. 1934. London: J. & A. Churchill. 10s. 6d. net.

**Manipulative Treatment for the Medical Practitioner.** By T. MARLIN, M.D., Ch.B., D.P.H., R.C.P.S. (Eng.), D.M.R.E., Medical Officer in Charge of the Massage, Electro-therapeutic, and Light Departments, University College Hospital, etc. Demy 8vo. Pp. 133 + vii, with 86 illustrations. 1934. London: Edward Arnold & Co. 10s. 6d. net.

**Surgical Anatomy and Physiology.** By NORMAN C. LAKE, M.D., M.S., D.Sc. (Lond.), F.R.C.S., Senior Surgeon, Charing Cross Hospital, etc.; and C. JENNINGS MARSHALL, M.D., M.S. (Lond.), F.R.C.S., Surgeon, Charing Cross Hospital, etc. Demy 8vo. Pp. 888 + x, with 238 illustrations. 1934. London: H. K. Lewis & Co. Ltd. 30s. net.

**Aids to Operative Surgery.** By CECIL P. G. WAKELEY, D.Sc. (Lond.), F.R.C.S. (Eng.), F.R.S. (Edin.). Second edition. $6\frac{1}{4} \times 4$ in. Pp. 225 + viii, with 3 illustrations. 1934. London: Baillière, Tindall & Cox. 3s. 6d. net.

**Abscess of the Brain: its Pathology, Diagnosis and Treatment.** By E. MILES ATKINSON, M.B., B.S. (Lond), F.R.C.S., Jacksonian Prize Essayist, R.C.S., 1926, etc. Medium 8vo. Pp. 289 + x, with 45 illustrations. 1934. London: Medical Publications Ltd. 21s. net.

**St. Bartholomew's Hospital Reports.** Edited by LORD HORDER, R. G. CANTI, W. SHAW, C. F. HARRIS, H. H. WOOLARD, R. C. ELMSLIE, W. G. BALL, G. EVANS, and J. P. ROSS. Vol. LXVI. Demy 8vo. Pp. 363 + xxiv. Illustrated. 1933. London: John Murray. 21s. net.

**Diathermy in General Practice.** By ERIC PAYTEN DARK, M.C., M.B., Ch.M (Syd.), late Radiographer, Royal Prince Alfred Hospital, Sydney. With special chapters by F. A. MAGUIRE, C.M.G., D.S.O., M.D., Ch.M. (Syd.), F.R.C.S., F.R.A.C.S., and GUY P. O. PRIOR, L.R.C.P., M.R.C.S. Second edition. Demy 8vo. Pp. 219 + xvi. Illustrated. 1935. Sydney: Angus & Robertson Ltd. 17s. 6d. net.